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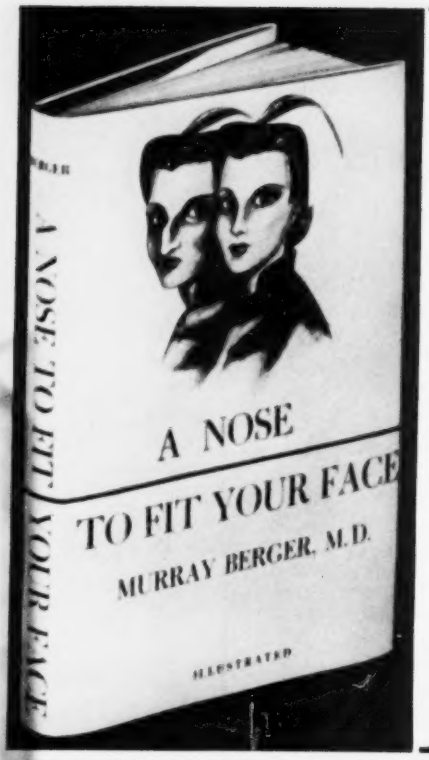

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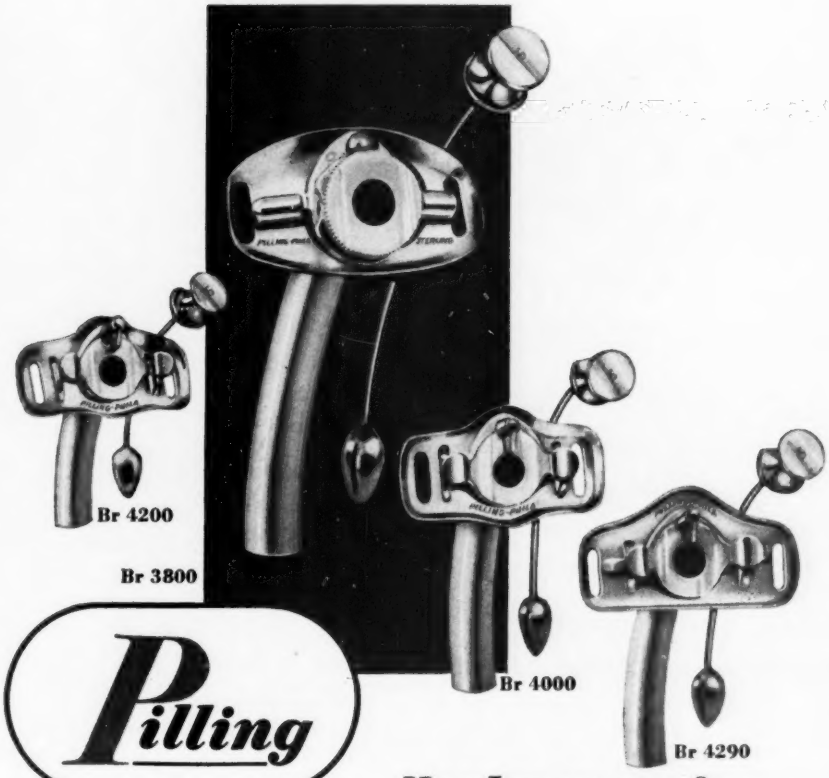

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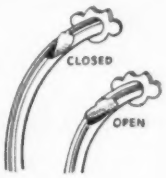

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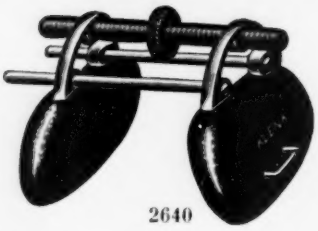

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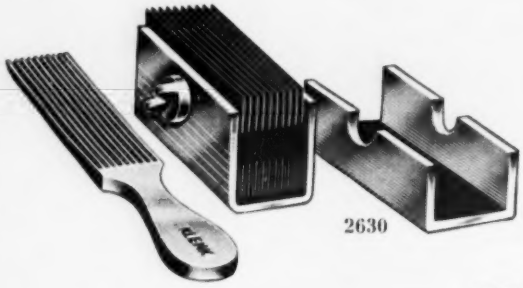

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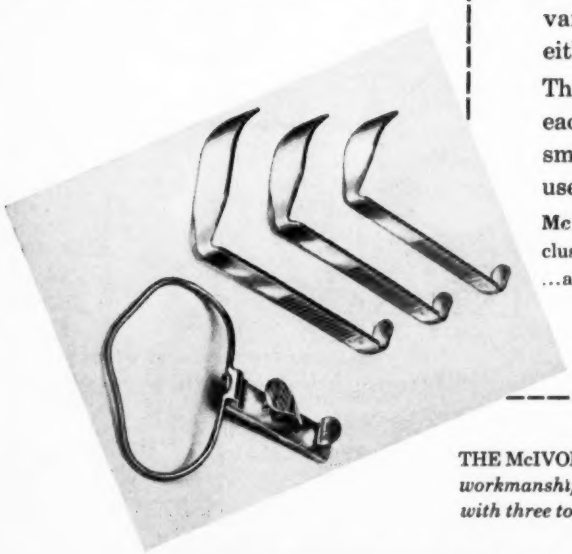

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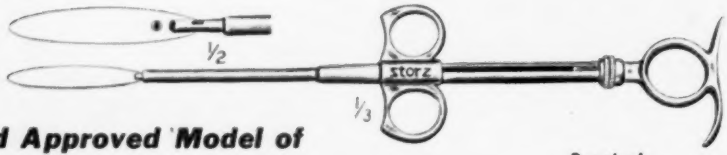

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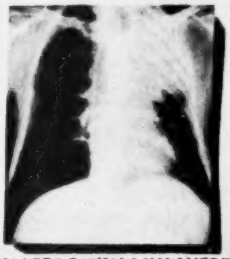

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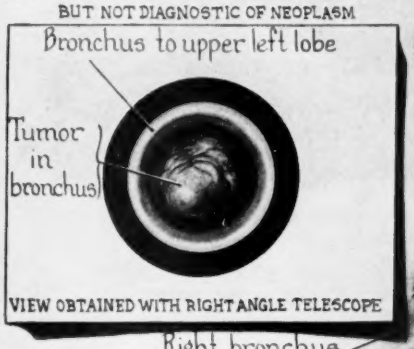

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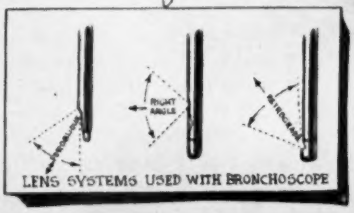

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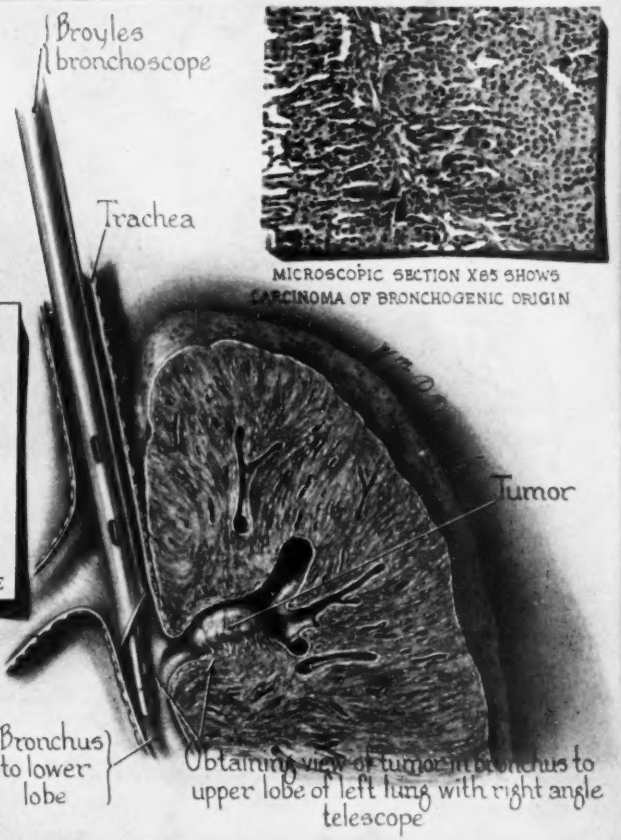
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VOLUME 55 JUNE 1952 NUMBER 6

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SCOPE OF OTOLARYNGOLOGY IN OCCUPATIONAL TRAUMA AND DISEASE

ABRAHAM GOLDNER, M.D.
FLUSHING, N. Y.

ACCIDENT and disease take an ever-increasing toll in American industry. That the average otolaryngologist sees few of these casualties during a routine practice is due not to the paucity of such cases but, rather, to a failure to appreciate their extent and importance. Occasionally a physician may be reluctant to engage in the paper work and litigation which are necessary to the proper handling of such cases. On the other hand, he has a definite duty to these patients, so that they will not be deprived of benefits due them, as he has also a similar obligation to facilitate the work of those who administer workmen's compensation laws.

The purpose of this presentation is to present briefly the legal background of occupational cases falling within the scope of the otolaryngologist and also to illustrate the extent and types of such cases together with the problems that they may present.

The therapeutic management of injuries and diseases resulting from occupation does not differ from the management of the same conditions found in everyday practice, but there are important features which bear emphasizing. The physician has a very clear-cut responsibility to the employer to provide the best of medical care, and his results are usually subject to critical review at various levels. He should keep accurate and complete records and provide both the employer, or his insurance carrier, and the workmen's compensation board with prompt clear reports.

While in ordinary cases we may permit ourselves the luxury of ignoring etiology, except where it is important to treatment, in occupational cases the question of etiology is often paramount, often transcending treatment in importance. The physician should not depend upon unproved concepts of disease, which have the unhappy faculty of thriving in our literature, but should analyze each case on its individual merits. And while it is necessary to possess a healthy skepticism in many problems, official referees do not expect the accuracy and validity of the research laboratory, except where such work actually exists and is applicable to the particular problem at hand.

Practically all the states of the union provide some type of protection for injured workmen. Naturally there are variations in the type and extent of benefits. New York State has been a pioneer in the provision and extension of benefits for injured workmen or those who may suffer disease as a result of their occupations.

The federal goverment provides similar benefits for injuries or disease sustained aboard vessels or similar places of employment.

Inasmuch as the cases presented come within the scope of New York State law, the legal basis of benefits as it affects the specialty of otolaryngology will be that of

this state. For variations of these benefits, the appropriate state agency would be pleased to provide detailed information.

COMPENSABLE OTOLARYNGOLOGICAL DISEASES AND INJURIES

There are five large classifications of injury or disease under which New York State extends benefits as it affects the specialty of otolaryngology. They are as follows:

I. *Accidental Injuries.*—This classification encompasses by far the greatest number of cases.

The law specifically states that compensation shall be payable for injuries or death incurred by employees in certain employments, and an extensive classification describing hundreds of occupations and types of employment is included.[1] In this group fall obvious contusions, lacerations, and fractures. Not so obvious, but still considered as accidental injuries, are conditions resulting from exposure to extremes of temperature, pressure, or irritating fumes.

II. *Loss of Hearing.*[2]—The law here provides for a defect the evaluation of which is exclusively the province of the otologist, and it states: "In case of disability partial in character but permanent in quality the compensation shall be 66 ⅔ per centum of the average weekly wages. . . . compensation for the complete loss of hearing of one ear for 60 weeks, for the loss of hearing of both ears, for one hundred and fifty weeks."

This loss of hearing may be due to many causes such as head trauma, occupational disease, and specific infection of the middle ear, as a result of either direct trauma or secondary infection. This subject will be further clarified under different headings.

III. *Occupational Disease.*[3]—Occupation is responsible for a considerable portion of disease which comes either directly or indirectly within the scope of the otolaryngologist. In New York State, the protection is broad, ranging from specific diseases to any or all diseases that can be shown to be due to work.

The following diseases or conditions which can involve the ear, nose, and throat are specifically noted: anthrax; zinc poisoning and its sequelae; poisoning by carbon disulfide or its sequelae; poisoning by nitrous fumes or its sequelae; poisoning by formaldehyde; chronic ulceration; glanders[4]; epitheliomatous cancer or ulceration of skin due to tar, pitch, bitumen, mineral oil, paraffin, or any compound product or residual of these substances; compressed air illness or its sequelae; exposure to fumes of sulfuric, hydrochloric, and hydrofluoric acid; respiratory disorders due to contact with petroleum products and their fumes; dermatitis venenata. In addition, Paragraph 29 of this section of the law includes the statement, "any and all occupational diseases" arising in all of the many employments listed in the law.

Under the latter section a host of respiratory allergies and conditions due to industrial dusts and environments become properly compensable disorders; to list a few: baker's asthma, asthma, vasomotor rhinitis of furriers, chronic nasal catarrh

1. Workmen's Compensation Law; New York State Workmen's Comp. Bd., July, 1949, p. 6.
2. Workmen's Compensation Law; New York State Workmen's Comp. Bd., July, 1949, p. 51.
3. Workmen's Compensation Law; New York State Workmen's Comp. Bd., July, 1949, p. 18.
4. Workmen's Compensation Law; New York State Workmen's Comp. Bd., July, 1949, p. 52.

due to excessive dust, and even hoarseness in salesmen as a result of excessive voice use.

IV. *Facial Disfigurement.*[4]—The law for this type of condition states:

1. The Board may award proper and equitable compensation for serious facial or head disfigurement not to exceed three thousand five hundred dollars, including a disfigurement continuous in length which is partially in the facial areas and also extends into the neck region as described in paragraph two hereof. 2. The Board, if in its opinion, finds the earning capacity of an employee has been, or may in the future be impaired, may award compensation for any serious disfigurement in the region above the sternal clavicular articulations anterior to and including the region of the sternocleido muscles on either side. . . .

This anatomical delineation quite obviously places a great many disfigurements within the field of otolaryngology. The addition of plastic techniques to the armamentarium of the ear, nose, and throat physician puts him in a position not only to evaluate better but to alleviate many of these disfigurements. The commonest condition requiring attention is fractured nose; however, facial fractures, injuries to the auricles, facial paralysis, industrial dermatitis, burns, and lacerations also come within the provisions of this section of the law.

V. *Aggravation of Disease and Consequential Conditions.*—The workmen's compensation law recognizes that disease may be aggravated by trauma. The proper allocation of responsibility will tax the acumen of even the most experienced physician. These cases comprise those of such conditions as otitis media following a contusion of an admitted preexistent infection, superimposition of traumatic or occupational deafness upon prior nerve or conductive deafness, and aggravation of a chronic sinusitis.

Consequential conditions refer to disability or illness arising out of a prior disability or from the treatment of an established condition. To illustrate: Deafness following use of streptomycin for an infection elsewhere in the body would be compensable. Similarly, a fractured leg resulting from a fall due to ataxia from a head injury would also be compensable.

Before proceeding to a discussion of actual cases as they occur, it would be worth while to describe disability from an occupational viewpoint. A medical report without an expression of the patient's disability would be incomplete. Disability may be classified as temporary, total or partial, or permanent, total or partial. The fact that a patient continues at work does not mean that he does not have a partial disability. For example, a patient with a persistent annoying tinnitus may continue working and yet from time to time be incapacitated from noisy work.

The next portion of this article will be devoted to a presentation of the clinical aspects of this problem. Where possible, actual cases will be presented to show the special problems involved. Although controversial cases will be presented, for the most part no discussion will be offered, inasmuch as some of these are extensive topics in themselves and it is our purpose to give a survey of the field rather than particular discussion on any one subject.

For convenience, the material will be presented in anatomical divisions except for a few subjects such as facial paralysis and allergy.

OCCUPATIONAL DISEASES AND INJURIES OF THE AURICLE

As an exposed appendage the auricle is quite susceptible to trauma. Lacerations and loss of tissues are the commonest affections. Burns, frostbite, and dermatitis

are other, less common, conditions. From an occupational standpoint the main problem is usually one of nominal disability and finally facial disfigurement. From a medical standpoint one can offer these patients the benefits of plastic surgery where indicated, for the most part with good results. Quite frequently a relatively trivial injury to the auricle will focus attention on the ear and hitherto unsuspected deafness will be demonstrated. This phenomenon occurs also in other types of injuries to the ear areas and will require careful evaluation in each case.

The following summaries of actual cases illustrate some of the preceding problems.

CASE 1.—C. R., while shoveling snow, sustained a frostbite of the right ear. There was no hearing loss, but he received an award because of a permanent deformity of the auricle.

CASE 2.—F. L. fell down steps, lacerating the left auricle; after the fall he complained of deafness and stuffiness of the left ear. An examination revealed the following positive findings: unequal pupils with a positive Wassermann reaction, chronic sinusitis, deviated septum, and mild bilateral obstruction of the Eustachian tubes. The Rinne test on the left was negative, and the caloric examination and x-rays indicated no abnormality. The audiogram demonstrated a 70.6% loss of hearing on the left and 29.6% on the right, as follows:

	Frequency, Cps						
	128	256	512	1024	2048	4096	8192
Right (db.)	30	25	25	25	40	75	70
Left (db.)	45	45	60	60	75	0	0

The obvious facial disfigurement is a simple problem, but more complex will be the proper allocation of responsibility for the deafness which the patient denied that he had had before this accident.

OCCUPATIONAL DISEASE AND TRAUMA OF THE EXTERNAL AUDITORY CANAL

There is a variety of diverse conditions which the otolaryngologist may encounter in the external auditory canals of workers. These vary from simple cerumen in the canal to facial paralysis as a result of involvement of the facial nerve as it courses through the osseous portion of the canal.

A simple contusion of the ear or a violent motion of the head may dislodge impacted cerumen or change its locus so that it produces symptoms. Generally, simple removal will solve both the compensation and the clinical aspects of this type of case. However, occasionally a similar type of patient may be found to be suffering from deafness of various types or a pathologic condition in the middle ear. Many of the latter patients are not aware of the condition and genuinely believe that the simple contusion precipitated their complaints. Once again a very complete examination and appraisal will be necessary, and the differential diagnosis should be clearly stated.

A condition very frequently encountered is minor burns of the canal in welders or burners. Unless complicated by infection they rarely present any problem. Occasionally a worker may appear with a discharging ear and give a history of sustaining a burn of his ear canal. As is common with many people ignorant of the progressive nature of disease, he will attribute his condition to the physical phenomena most proximate to it, a sort of *post hoc, ergo propter hoc* line of reasoning frequently encountered and, unfortunately, accepted in compensation practice. It should be remembered at this time that burns are not caused by the myriad incandescent sparks which shower these workers except at close contact. The temperature

and size of a spark will preclude it from causing a burn in the canal, which is usually well protected by a layer of cerumen or hair at the external canal acting as a guard. A severe burn will generally have a particulate piece of metal as the source which has to be removed from the canal.

Industrial dermatitis of any type may extend from the auricle to the canal, and the problems presented will be similar to those in which the condition is present exclusively in the auricle.

External otitis, with or without fungous infection, is seen frequently among life guards, swimming instructors, and, more recently, water ballet performers. In these groups the condition can be properly classified as an occupational disease and may cause long periods of disability.

Lacerations involving the membranous and cartilaginous canal may result in varying degrees of stenosis but of themselves rarely cause deafness.

The following is an example of a burn of the external canal and auricle, with subsequent complaint of deafness.

CASE 3.—J. P. burnt his face, right auricle, and canal with hot grease. Three weeks after the accident he noticed deafness and tinnitus of the right ear. Positive findings one year after the accident were a scar of the right external canal, a normal drum membrane, and a postnasal discharge; Rinne tests on both sides were negative, the audiometer showed a 12% loss of hearing in the right ear and 10% in the left. The case was closed with a finding of unrelated deafness and a facial disfigurement.

In the next case a burn of the canal and drum was caused by a hot metal chip. The external otitis was overshadowed by otitis media, which resulted in a permanent hearing defect.

CASE 4.—A. T.'s ear was injured by a hot metal chip which flew into his ear canal. This resulted in extensive external otitis and an otitis media. The canal healed rapidly, and the ear drum healed in about six months. One and one-half years after the accident he was awarded compensation for a 11% loss of hearing in the left ear.

The following is an illustration of fracture through the anterior canal wall with derangement of temporomandibular articulation and loss of hearing. X-rays did not show the fracture, inasmuch as the involved area is made up of cartilage and fibrous tissue and is very susceptible to trauma.

CASE 5.—L. S. struck his chin on the edge of a table. After this, he experienced pain in both ears, with tinnitus in the right ear and bleeding from the right canal. An examination revealed a bulging of the right glenoid fossa in the anterior canal wall, with bleeding. The drum was intact. Repeated special x-ray studies of the mastoid and right temporomandibular articulation were reported as showing no abnormalities. Audiograms revealed a loss of hearing of 20% in the right ear and 19.5% in the left ear.

OCCUPATIONAL DISEASE AND TRAUMA OF MIDDLE EAR, MASTOIDS, AND ADJOINING TISSUES

The middle ear cavity, mastoid cavity, and Eustachian tubes are continuous anatomically and disease of one usually affects the other two. Meningitis most frequently represents a complication of mastoiditis; therefore, conditions affecting these structures will be discussed together. It is also convenient to discuss the consequence of injury to these structures in two large groups: (1) purulent otitis media and its complications, and (2) nonsuppurative otitis media.

1. *Purulent Otitis Media and Its Complications.*—This condition is commonly seen in industrial practice. It most commonly occurs in the following conditions.

(*a*) Skull Fractures: Fractures of the temporal bone are often accompanied by injury to the middle ear and its components. Laceration of the tympanic membrane is common, although surprisingly few cases develop into full-blown purulent otitis media. X-ray demonstration of fractures of the temporal bone, particularly of the base, is difficult and is often overlooked, inasmuch as the ear condition is often overshadowed by the injury to brain tissue. It is not necessary that the injury be to the affected side, nor is it necessary that an actual fracture be demonstrated. However, in the vast majority of cases the trauma is severe enough to produce unconsciousness. If a purulent otitis media develops, then, of course, it can progress to mastoiditis or meningitis or even brain abscess. Such conditions are often late complications of the fracture. A break in the continuity of the bone, healing as it does by fibrous union, can also act as a potential channel of meningeal invasion long after the orginal injury has been treated and adjudicated.

(*b*) Explosions and Blasts: Experience during the war years and during civil disasters, such as the Texas City catastrophe,[5] has confirmed the susceptibility of the ears to injury. Any variety of tympanic laceration may occur. As in head injuries, there is a low incidence of suppurative complications. Of greater significance is temporary and permanent damage to the organ of Corti with resultant loss of hearing.

(*c*) Direct Injury to Tympanic Membrane: Owing to the anatomical placement of the tympanic membrane and angulation of the external canal, hair, and cerumen, direct injury is not common. Welders will occasionally present themselves with a direct burn of the tympanic membrane. Such injuries are caused by large pieces of particulate molten metal and will often burn the canal simultaneously. The perforations are apt to be extensive and prove difficult to close. In agricultural workers, occasionally a twig will cause a perforation of the membrane; these perforations usually heal promptly without incident. In other types of workers protruding wires may also cause similar injury.

(*d*) Extension of Disease from External Canal: As in nonoccupational disease, the middle ear can become involved from direct extension of a process in the external canal. This condition is seen frequently in those whose occupation involves swimming, such as lifeguards and camp counsellors. It also may be seen in welders as an extension of the secondary infection incident to burns of the canal. It may not be recognized until the swelling of the canal has subsided sufficiently to permit inspection of the middle ear.

(*e*) Extension from Nasopharynx: Although the exact mechanism is not known, it is commonly recognized that an infection of the nose and throat may extend to either ear. Thus, when any such condition is found to occur as the result of occupation or trauma, the resultant otitis media must also be considered as related. This condition is seen most frequently in lifeguards and divers, the group mentioned in the previous category.

5. McReynolds, G. S.; Guilford, F. R., and Chase, G. R.: Blast Injuries to the Ear: The Texas City Disaster, Arch. Otolaryng. **50**:1 (July) 1949.

(*f*) Aggravation of the Preexisting Otitis Media: This group of cases is the most difficult to evaluate from the standpoint of causal relationship of the active process and any permanent damage to hearing that may ensue. In the previous category of swimming occupations such aggravation is common and is obviously related to occupation. After severe head injuries or contusions in the region of the ear, it is not uncommon for a quiescent otitis media to become active. This activity, however, should occur within a few days after the trauma if it is to be considered related. It is not unusual to have an old inactive otitis media without knowing of it. Thus, frequently when such processes become active after a trivial trauma, or if the patient is working in a noisy environment, such as a shipyard, it will be attributed to such factors. Inspection of the ear drum and x-rays of the mastoids should indicate quite clearly the age and nature of the process, and, in the absence of the previously described causative factors, such activity is not related to either the imagined trauma or the occupation. From a compensation viewpoint, apart from temporary disability, in the course of otitis media and its complications, it is important to indicate when the condition became permanent and the amount of hearing loss attributable to the condition. In otitis media, it is difficult to fix a dogmatic time that can be considered as the time when the process has become finally quiescent. However, an ear that has not discharged for one year can be considered to be in an inactive phase. Yet no matter when such activity may recur, its treatment and disability would have to be considered as part of the original picture. In aggravation of a preexistent otitis media, it will be necessary to fix such time if possible when the process has returned to its anteaccident status. The deafness in these cases varies in degree, and is usually of mixed perceptive and conductive type.

2. *Nonsuppurative Otitis Media.*—In this group are included cases of conditions that may be also diagnosed as catarrhal otitis media, acute or chronic, Eustachian catarrh, or aerotitis media. It has become common practice to diagnose as catarrhal otitis media any deafness accompanied by retraction of the membrane, with or without evidence of chronic tonsillar or nasal infection and with or without appearance of blockage to air of either Eustachian tube. Variations in texture and inclination of the tympanic membranes exist without auditory complaint or deafness. When nasal obstruction or infections produce impairment of hearing, it is usually of the conductive type. Tonsillitis is rarely, if ever, a factor in deafness. Evaluation of the patency of the Eustachian tubes is not always an exact procedure, and the information gleaned from standard procedures does not always reflect the role of the Eustachian tubes in the condition complained of. Diagnosis should be carefully made, and experience shows that in the absence of otosclerosis, the classic triad of lowering of the threshold for low tones, reversal of ratio of air to bone conduction, and increase in bone conduction remains a dependable standard for diagnosis. Nonsuppurative otitis media as an occupational complaint occurs most frequently in persons engaging in the following types of occupations or with the type of injury mentioned.

(*a*) Occupations Involving Changes in Barometric Pressure: The literature of recent years contains references to what has been called aerotitis media in aviators and other flying personnel. A similar condition is often noted in "sandhogs" and divers. In the "sandhogs" the clinical picture may be confused because of the presence of coexistent high-tone deafness as a result of noise or of a nerve deafness

due to embolic effects of released blood stream nitrogen, such as occurs during attacks of the "bends."

(*b*) Concussion of the Middle Ear: An exudative reaction may occur in the middle ear after blows in the area and sudden exposure to loud noise such as telephone operators are subjected to. This condition may also be accompanied by effects of cochlear damage.

In the next case purulent otitis media developed as a complication of skull fracture; mastoiditis then developed, and finally death occurred at a much later period from meningitis.

CASE 6.—J. L. was struck in the head by a heavy oak beam, was unconscious for 10 days, and complained of dizziness, tinnitus, headache, and deafness of the right ear. An x-ray of the skull showed a depressed fracture involving the right parietal bone, the orbit and also the floor of the anterior fossa and the right sphenoid bone. An ear, nose, and throat examination performed several months later showed scarring and retraction of both drum membranes and severe deafness of the right ear, and upon testing, the right labyrinth showed no reaction. Two years after the original accident, the patient lapsed into a coma and died. An autopsy revealed a fracture extending into the greater wing of the sphenoid and leptomeningitis involving the meninges of the anterior and middle fossa.

This case illustrates the importance of careful otologic examination and follow-up and, more importantly, it illustrates that even years following a head injury with fracture of the petrous bone or sinuses, a delayed leptomeningitis with abscess formation may result in death.

In the following case otitis media occurred as an extension of otitis externa.

CASE 7.—A. T. sustained a burn of the external canal as the result of being hit with a piece of hot slag. The canal was severely excoriated and inflamed, and several days following the accident the middle ear became inflamed, and a perforation developed spontaneously in the central portion of the tympanic membrane. Ultimately the otitis externa and the otitis media healed, but the tympanic membrane was scarred and thickened and there was a mild permanent deafness.

This case illustrates that occasionally the tympanic membrane may be injured directly by a wire.

CASE 8.—W. O., while using a wire to clean a beer drain, accidentally caused the wire to enter his left ear canal and sustained a perforation of the left drum membrane, with bleeding. There was no disability and the only residuum in this case was a faint scarring of the tympanic membrane which healed without complication and without hearing loss.

In the next case middle ear disease resulted from extension of infection via the Eustachian tubes.

CASE 9.—A. D. is a life saver, and in the course of his employment it has been necessary for him to dive frequently under water. Positive physical findings consisted of a chronic nasal catarrh, a mixed deafness, and tubal obstruction. He was treated by the usual measures of inflation and irradiation and was left ultimately with a mild deafness.

The following case shows the aggravation of a preexistent chronic purulent otitis media with development of mastoiditis and that five years after the case was closed, right-sided paralysis and aphasia developed; these were found to be due to a temporal lobe abscess.

CASE 10.—O. A. fell off a ladder, sustaining a fracture of his left little finger and an injury to his head. Discharge, impaired hearing, and pain developed eight days following the accident. X-rays showed cholesteatoma and sclerosis of the left mastoid, and local examination of the

middle ear showed a chronic perforation with discharge. Eventually a causal relationship was established on a basis of aggravation of a preexistent chronic mastoiditis and otitis media, and a radical mastoidectomy was performed. This was complicated by a surgical left facial paralysis. Five years after this case was closed, sudden paralysis of the claimant's right arm and leg and an aphasia developed. Operation disclosed a temporal lobe abscess.

This case not only illustrates aggravation of a preexistent chronic otitis media but shows that, as in Case 6, many years following the closing of such cases serious complications may occur as the result of reactivation or extension of a disease process in the mastoid bone.

The following is an example of the development of a chronic catarrhal otitis media with subsequent deafness as the result of exposure to changes in pressure.

CASE 11.—C. F. had been a "sandhog" for 27 years. In the course of his work it was necessary for him to work under conditions of variable pressure and to go through the various processes of compression and decompression to prevent the development of "bends." He suddenly experienced stuffiness in both ears and marked tinnitus while going through the decompression chambers on his way to his job. An examination showed marked retraction of both drums, and there was a severe conductive deafness in both ears.

This next case illustrates the destructive effect of hot metal or slag on the tympanic membrane and the chronicity of the resultant perforation and infection.

CASE 12.—C. N., while cutting metal in a combustion chamber, got a piece of molten steel in his left ear. Initially a vesiculation and a burn of the tympanic membrane and canal were noted; these were followed by an extensive perforation. Two years after this incident the perforation was still present in the left tympanic membrane and there was no active otitis media, but there was a permanent loss of hearing amounting to about 45%.

OCCUPATIONAL DISEASE AND TRAUMA OF THE INNER EAR

The bony shell which encases the labyrinth and cochlea has proved to be an effective barrier to the accurate study of these most important sensory organs. However, it has not shown itself to be as effective a protection for these structures against the effects of trauma or occupational disease. The main symptoms that are caused by disease of the inner ear are deafness and tinnitus. Occasionally dizziness and pain may also be complained of.

The conditions that can occur in the inner ear of workmen may be subdivided into two large groups. Deafness and tinnitus following head injuries of varying degrees form a substantial group. In the same category are similar effects arising as a result of exposure to sudden extreme loud noise or explosions. A much larger group may be classified under the heading of chronic occupational deafness, and in this group we have the main classification due to the effects of harmful prolonged noise. We also have harmful effects as the result of industrial poisons and effects upon the inner ear due to caisson disease.

Disease of the Inner Ear Following Acute Trauma.—There is almost a classic otological picture following petrosal fractures. It consists of deafness of varying degrees mainly affecting the higher tones and a nonfunctioning labyrinth. Indeed, these findings following head injury will often point to the correct diagnosis of a basal skull fracture when roentgenography of these areas is noncontributory to the diagnosis, because it is quite difficult to pick out a fine fissure fracture of the petrous pyramid from the many overlying shadows and structures seen by x-ray in this area. The effect on hearing is maximal at the time of injury, and the

hearing tends to improve as time goes on. It does not become worse. It is not necessary that the labyrinth be damaged to the extent that vestibular examination shows complete loss of response. If the vestibule has been damaged, such damage is not necessarily permanent. It is not necessary that the head trauma be accompanied by definite unconsciousness. Severe blows causing momentary stunning can transmit a harmful force to the cochlea, leading to varying degrees of hearing impairment. Experience has shown that in the elderly, the effects upon hearing are apt to be severer, probably because of increased fragility of the tissues. Traumatic deafness may be superimposed upon preexistent deafness of the perceptive, the conductive, or the mixed varieties, and in the absence of accurate auditory data prior to the accident an evaluation of causally related traumatic hearing loss is difficult but is always possible.

CHRONIC OCCUPATIONAL DEAFNESS

In 1945, I wrote a comprehensive article in which the importance and scope of the subject of chronic occupational deafness was outlined.[6] Since that time a number of additional reports have accumulated in the literature, and indeed the problem of occupational deafness has proved to be so extensive and important that it has become the subject of additional research and conferences at a national level.

This type of deafness may be subclassified into (1) deafness due to excessive noise, (2) deafness caused by poisons and chemical agents, and (3) deafness due to changes in barometric pressure.

In a general way, the following occupations can be classified as noisy, to a degree that in workers who work at such occupations deafness may develop as a result of exposure to excessive noise. Employment in all phases of the metal industries, including the various processes attendant on the fabrication and shaping of metal, is among the prime offenders. Employment in the shipyards has proved to be harmful to hearing, particularly for those workers who either are directly concerned in the fabrication of metal or perform duties that make it necessary for them to work in the vicinity of such processes. Any industry in which pneumatic tools are employed also may cause hearing loss. Railroad workers and workers in the spinning and weaving industries, particularly where the machines are noisy and vibrate excessively, may experience loss of hearing. Telephone workers or switchboard operators may also suffer from loss of hearing. In such cases, the loss may be due to sudden exposure to loud unpredictable sounds, directed at maximum intensity to the ear.

Industrial poisons, such as quinine, lead, tobacco, and arsenic, may cause impairment of hearing. Other chemicals, such as phosphorus, carbon disulfide, and paraphenylene dyes, may also cause cochlear degeneration. One must now also include as cases of deafness indirectly caused by industry those cases in which streptomycin has been administered, with resultant loss of hearing.

In the aviation trade, aviators may suffer from hearing loss due to the effect of pressure changes. Workers and mechanics who are exposed to the noise of airplane engines, and now the more extensive noise of jet engines, also may suffer

6. Goldner, A. I.: Occupational Deafness, with Special Reference to Chronic Occupational Deafness, Arch. Otolaryng. **42**:407 (June) 1945.

hearing loss. Caisson workers may also suffer hearing loss resulting from recurrent catarrhal otitis media or due to embolic effect on the blood supply of the cochlea.

The diagnosis of occupational deafness, particularly the uncomplicated type, should be fairly simple. The criteria that I enumerated in 1945 have withstood the test of experience and many examinations since that time. Briefly, these criteria are as follows:

1. The impairment is usually bilateral unless one ear has been exposed exclusively.
2. The subject is usually middle-aged or older, unless exposure has been extreme.
3. The tympanic membrane is normal. In this connection, differences in texture and color of the tympanic membrane are of minor significance.
4. Bone conduction is diminished in relation to air conduction, indicated in otologic terms as a positive Rinne test and a positive Schwabach test.
5. The audiograms show a classic pattern with normal or moderately diminished low tones and excessive diminution of higher tones. In early cases, the 4096 db. frequency shows a maximum drop.
6. The vestibular responses are normal.
7. Usually the Eustachian tubes are patent, but transitory blocking of the Eustachian tubes is of no significance unless accompanied by low-tone hearing loss and increased bone conduction in relation to air conduction.

Chronic occupational deafness may and frequently does coexist with other types of deafness.

In older workers, some allowance would have to be made for the high-tone drop occasionally seen past the age of 50. Such a drop is not constant and depends to a large measure upon the hereditary background of the patient, and the extent of cerebral arteriosclerosis. In cases of prolonged employment, it is quite likely that the high-tone drop due to excessive exposure to noise will antedate the problematic, occasional high-tone drop seen in people past the age of 50.

My experience with large groups of workers deafened as a result of exposure to noise has shown that persons with perforations of the tympanic membrane are more susceptible than others to the harmful effects of excessive noise. This increased susceptibility is based upon the loss of the protective action of the tympanic membrane and the intact ossicular chain. From a compensation standpoint, it becomes important to decide what part of the hearing loss is related to exposure to harmful noise.

Workers with chronic otitis media or more particularly with perforations of the tympanic membrane are very poor risks for employment in noisy places, and in no circumstances should be employed in such places.

Workers with conductive deafness are not as susceptible to the harmful effects of excessive noise and psychologically appear to tolerate such exposure better than the average person. However, they are not immune to harm, and presently there may be imposed upon a preponderant low-tone drop an additional high-tone drop beyond the 2048 cps frequency, which may accentuate and aggravate the auditory disability.

EVALUATION OF HEARING LOSS

It will not be possible within the scope of this paper to dwell upon the most important and neglected subject of evaluation of hearing loss. However, one cannot leave the subject of diseases of the ear without making some comments.

It is most disheartening to the scientific mind to observe the lack of correlation in estimates of hearing loss for the same patient when such estimates are performed

by several physicians. The American Medical Association standardization values for hearing loss is a valuable instrument. However, it must be properly applied, and the limitations of the technique must be recognized. Too many physicians, and particularly the lay people, look upon the audiogram as some sort of foolproof graph of an individual's actual hearing loss, not realizing that it represents only the subjective response of a subject to selected frequencies at a given moment and, like all subjective tests, is subject to a wide variation for a variety of reasons. The perception of pure tones is not necessarily related to the perception of conversation; this is particularly evident in patients who are illiterate or poorly educated or who speak foreign languages. These patients will often give better responses comparatively on the audiogram than is evidenced by an evaluation of the actual loss to conversation. Yet, if used and interpreted properly, the audiometer is the most valuable instrument for the evaluation and diagnosis of hearing loss. Repeated audiograms are a most valuable means of demonstrating malingering in partial deafness, inasmuch as when a process is stationary under similar conditions there should be not more than a 5 db. variation in response, and it would require an unusual human memory to duplicate response levels on separate occasions deliberately.

Conversational voice tests and whisper tests have only limited value. In my hands their most valuable function has been to obtain a general idea of the patients' responses and anticipate, if possible, exaggeration and malingering.

The tuning fork tests are still quite valuable in the evaluation and diagnosis of hearing disorders. For more accurate evaluation, it is suggested that the bone and air conduction be actually measured in seconds and so recorded. A negative Rinne test associated with a positive Schwabach test, indicating increased bone conduction, practically always indicates a conductive hearing loss. Similarly, a positive Rinne test, indicating better hearing by air than by bone, and a diminished Schwabach test, indicating diminution in bone conduction, almost always indicate a perceptive or nerve lesion.

A yardstick is needed that can be universally applied. Masking of the opposite ear should be universally employed when there is a substantial difference in hearing level, and it should be recognized that the simple plugging of an ear with a finger or other object does not materially reduce the level of auditory acuity. Failure to mask an ear which has substantially better hearing than the opposite ear will give rise to false interpretations and a completely false picture of the basic lesion.

In conclusion, the proper use and interpretation of conversational voice and whisper tests and the proper use and evaluation of tuning fork responses in conjunction with an intelligent interpretation of audiogram responses will give an accurate estimate of any patient's hearing loss and should in most instances give rise to a correct diagnosis. However, such factors as malingering, unrecognized deafness, perception of sound through a good ear, language difficulties, and masking by tinnitus will confuse the picture and should be taken into consideration at all times.

ILLUSTRATIONS OF TRAUMATIC AND OCCUPATIONAL DEAFNESS

The following case demonstrates the effect of head trauma upon the cochlea.

CASE 13.—A. R., a 46-year-old mechanic, was struck in the right occipital area by a heavy steel object. He was unconscious about 30 minutes, and x-rays revealed a fissure fracture

extending from the right occipital bone to the base of the skull. In addition to the usual complaints of headache and dizziness, he complained of tinnitus and impaired hearing of the right ear. The physical examination of the ears, nose, and throat disclosed nothing of significance. The results of the hearing examination are shown in Chart 1. Vestibular examination demonstrated loss of response to ice water in the right ear.

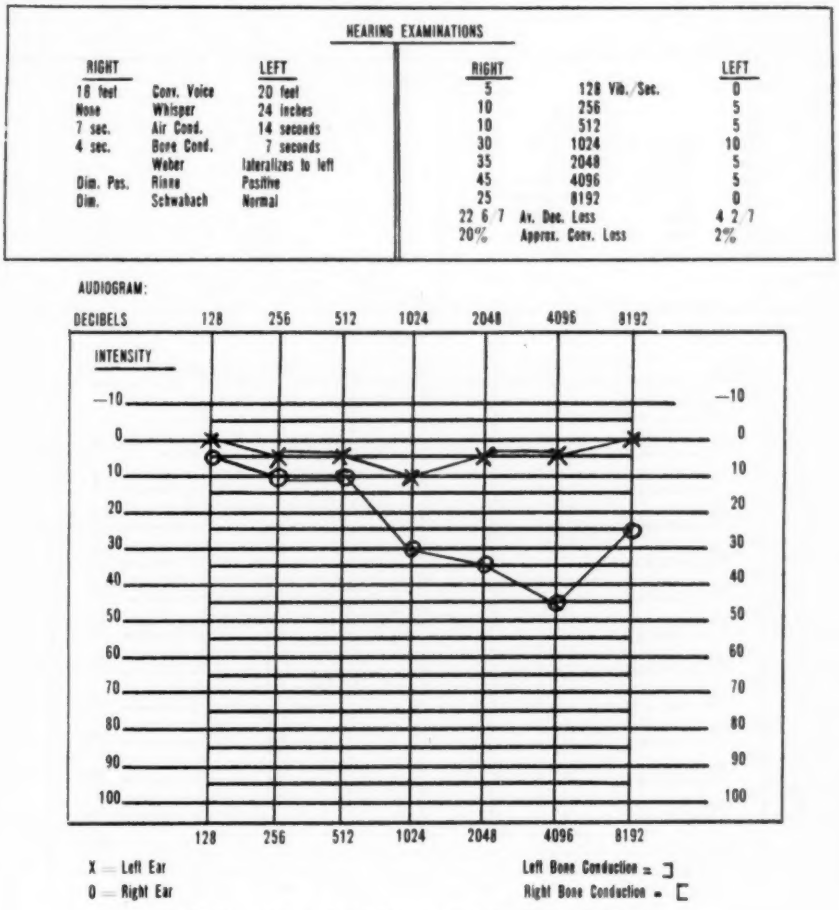

Chart 1 (Case 13).—Effect of head trauma on cochlea.

This case presents the typical finding in a classic case of petrosal fracture. It was ultimately closed by an award for partial loss of hearing of the right ear in addition to a lump-sum settlement.

The following is a case of classic occupational deafness due to excessive noise.

Case 14.—B. C., a 49-year-old chipper and caulker, has been doing this type of work in shipyards for 30 years. A good part of his work is performed in confined ships' holds. For some time prior to his examination, he noticed a gradual diminution of hearing bilaterally and also a constant annoying tinnitus. Except for the hearing examination, the ear, nose, and throat examination was completely without result. The picture as shown in Chart 2 is classical.

The following case presents an instance of deafness in a "sandhog."

CASE 15.—J. R., a 45-year-old laborer, has been working as a "sandhog" for the past 15 years. He has had several attacks of the "bends" and after several periods of decompression and compression has noted fullness of his ears and tinnitus. He also feels that his hearing is impaired. A physical examination revealed thickened bulging drums bilaterally in a state of chronic congestion. The Eustachian tubes were patent to direct inflation. The nose and throat were normal. The hearing examination (Chart 3) disclosed a combined deafness. The low-tone

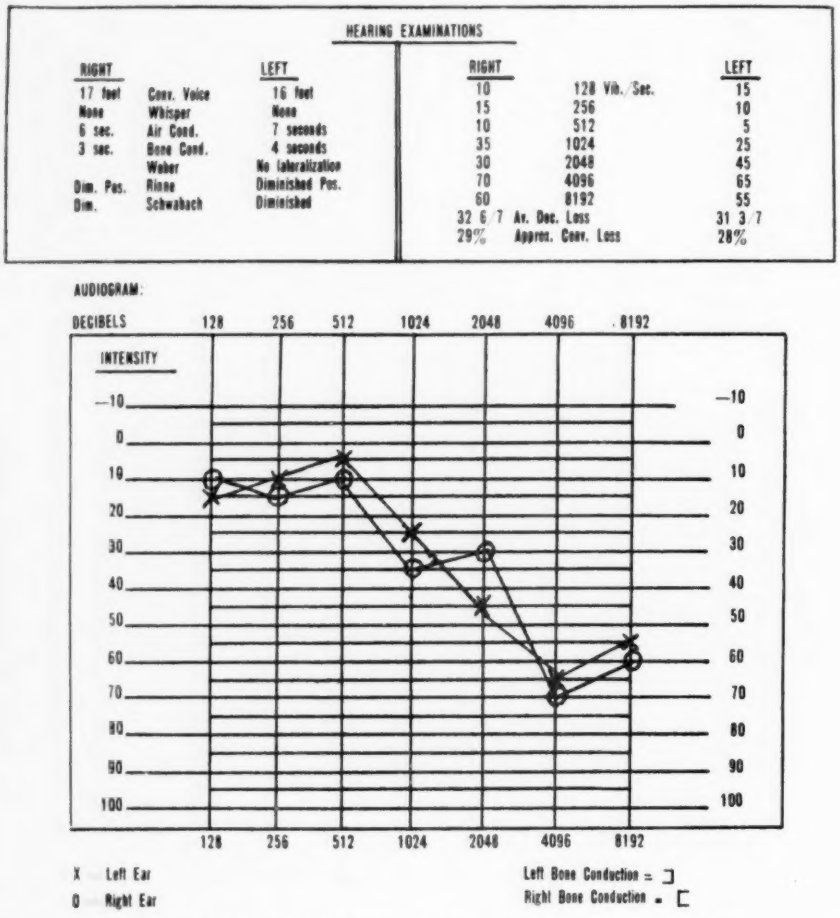

Chart 2 (Case 14).—Classical occupational deafness in chipper and caulker.

loss is due to chronic catarrhal otitis media as a result of changes in pressure. It is conductive. It is superimposed on a high-tone loss with perceptive characteristics. This is probably due to embolic phenomena in the cochlea, but it could also be the result of excessive confined noise.

FACIAL PARALYSIS IN WORKERS

On first consideration, the problem of facial paralysis does not seem to properly come within the scope of the otolaryngologist, and yet from the standpoint of the

anatomical course of the nerve exclusive of its origin in the brain it comes most properly within the scope of otolaryngology. In addition, there are problems in diagnosis, requiring simultaneous evaluation of hearing and vestibular function, in order to evaluate the extent of damage to the facial nerve and origin and location of the lesion; and finally, it is only by means of the plastic techniques developed in otolaryngology that any relief can be afforded to the victims of this disease with

HEARING EXAMINATIONS

RIGHT		LEFT
14 feet	Conv. Voice	12 feet
None	Whisper	None
3 sec.	Air Cond.	4 seconds
4 sec.	Bone Cond.	6 seconds
	Weber	No lateralization
Dim. Neg.	Rinne	Diminished Neg.
Dim.	Schwabach	Diminished

RIGHT		LEFT
40	128 Vib. Sec.	30
35	256	35
25	512	15
35	1024	30
50	2048	40
60	4096	50
50	8192	45
42 1/7	Av. Des. Loss	35
38%	Approx. Conv. Loss	32%

AUDIOGRAM:

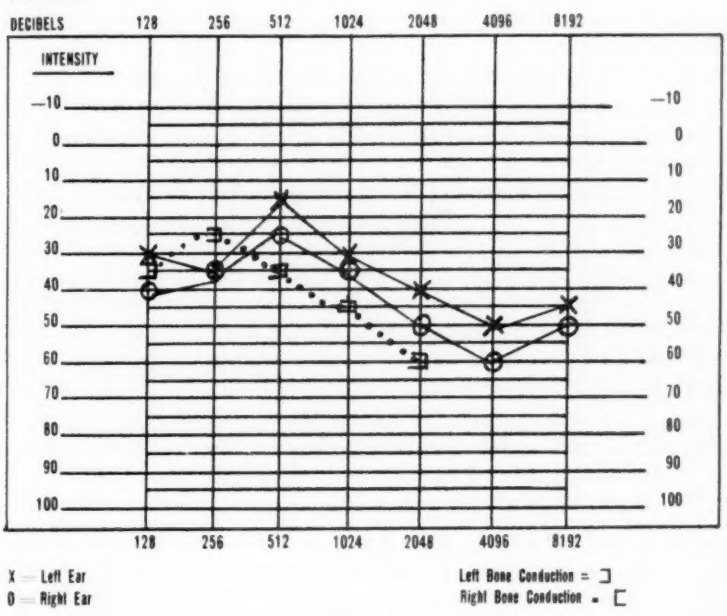

Chart 3 (Case 15).—Effect of barometric pressure change and bends.

permanent facial disfigurement. There are also cases in which prompt surgery of the facial canal may prevent the development of permanent full-blown Bell's palsy. There are two main varieties of facial palsy seen in industrial practice. By far the commonest is the so-called idiopathic type, in which exposure to a draft is alleged to be the precipitating factor. The other type is that seen following head injuries.

In the idiopathic type, laboratory work has confirmed the clinical observation that local chilling may produce facial palsy.[7] However, it should be borne in mind that facial palsy may occur in diabetes, syphilis, cranial neoplasms, cerebral vascular disease, and underlying otitis media. These conditions should be ruled out before local chilling is accepted as an etiologic factor. Fortunately, in the great majority of these cases the condition resolves completely with or without treatment. In a small proportion of the cases the patient is left with the extensive deformity of permanent facial palsy. If it were possible to anticipate this type of case, an early decompression of the facial canal might prevent the deformity.

Facial paralysis following head injuries does not offer any obscure problems of causal relationship. However, by careful examination it is often possible to locate the primary lesion. Generally, such paralysis occurs only in the severe types of fracture of the skull and is usually accompanied with hearing loss and vestibular damage. If the lesion occurs in the middle ear before the branches to the tensor tympanic muscles are given off, the patient will have, in addition to paralysis, hyperacusis and annoying tinnitus, while if the lesion occurs at the level of the Glaserian fissure where the chorda tympani is given off in the osseous external auditory canal, in addition to the paralysis, the patient may demonstrate absence of taste sensation on the dorsum and anterior two-thirds of the tongue, together with increased salivation.

Permanent facial paralysis, no matter what the cause, can be alleviated and helped by the restorative techniques of plastic surgery. The use of such techniques will often help to rehabilitate a patient and minimize the serious mental effects of serious facial disfigurement.

The following are a few examples of facial paralysis in workmen.

Case 16.—A sewing-machine operator stated that she was exposed to a draft from an open window and subsequently paralysis of the right side of her face developed. A physical examination disclosed nothing of significance except for the demonstration of complete right Bell's palsy. The patient lost only a few days from her work; she received physical therapy for about two months, and the palsy disappeared completely. A causal relationship was established between Bell's palsy and exposure to the draft.

Case 17.—A. W., an office worker, alleges exposure to the draft of a fan which was so placed as to blow on her continually. Physical examination revealed the presence of complete Bell's palsy but nothing else of significance. The claimant was treated for approximately two years and received all modalities of physical therapy. The paralysis persisted, and the case finally was settled with the maximum award for permanent facial disfigurement.

This case is one of the few in which such conditions go on to complete permanent paralysis, and it is possible that if an early decompression of the facial canal had been performed in this case the permanent paralysis would have been averted. In any event, failing such prophylaxis, the patient should have been offered the benefits of plastic surgery.

Case 18.—W. R. fell off a truck and sustained a fracture of the left occipital area. In addition to his head injury, left facial paralysis, disturbance of taste, loss of taste on the left side of the tongue, and mild impairment of hearing developed. The salient features of the otolaryngological examination were the presence of the impaired hearing with the high-tone drop, a normal labyrinthine reaction, and demonstration of loss of taste on the left side of the tongue. The facial paralysis ultimately improved, and the sole residuum was a slight impairment of hearing and complaint of tinnitus.

7. Kettel, K.: Bell's Palsy: Pathology and Surgery, Arch. Otolaryng. **46**:427 (Oct.) 1947.

This case illustrates the localization of injury to that portion of the facial canal in the region of the Glaserian fissure. It also indicates that when the vestibular reaction is normal the hearing impairment is likely to be minimal and that the lesion is below the internal auditory meatus.

CASE 19.—J. J. was struck on the back of the head by a box weighing 10 lb. (4.5 kg.), and one hour later a facial paralysis appeared on the right side. Examination revealed the presence of a right facial paralysis and impairment of taste on the right side of the tongue. It was felt that the injury caused the rupture of a blood vessel in the facial canal at the level of the chorda tympani nerve emergence. Most of the facial paralysis ultimately disappeared, without much in the line of specific therapy.

CASE 20.—A. S. fell 20 ft. (6 meters) and sustained a fracture of the right petrous apex. He was unconscious, and in a short time a right facial palsy developed. Examination also revealed severe loss of hearing of the right ear with a predominant high-tone loss and absence of the vestibular response. The palsy, the absence of the vestibular response, and the hearing loss were permanent and represented a substantial increase in disability over the temporary total and partial disability due to the effects of the skull fracture.

OCCUPATIONAL DISEASE AND TRAUMA OF NOSE AND SINUSES

The nose and, less often, the sinuses are frequently injured in the course of employment. Similarly, the nasal mucosa is susceptible to deleterious effects as a result of inhalation of noxious and irritant fumes and along with the remainder of the respiratory mucosa is extremely susceptible to sensitizing influences leading to debilitating and often disabling nasal respiratory allergy.

As in the auricle, and not peculiar to the nose itself, the skin of the nose and vestibule are susceptible to a variety of industrial skin afflictions. For the most part, these patients are seen by dermatologists, but occasionally they are seen by ear, nose, and throat specialists, so that it behooves such specialists to be aware of these conditions. From an occupational viewpoint, there are few problems. If scarring remains, there will be facial disfigurement. Good industrial housekeeping and the avoidance of irritants are the main points of treatment.

Injuries to the nose are among the commonest forms of facial accidents and form a considerable portion of those cases that come under the direct care of the otorhinolaryngologist. There are two features which require particular attention: facial disfigurement and adequate nasal ventilation. The adoption of rhinoplastic techniques by the average otorhinolaryngologist has done much to reduce the amount of facial disfigurement and to provide adequate nasal ventilation. It also serves to increase the scope of services to be offered by such specialists. Correction of nasal deformity should be offered to those injured workers who desire such correction.

This is particularly desirable for females and for young or middle-aged workers whose facial appearance is important. X-rays should be taken of all nasal injuries, not only for diagnostic but also for medicolegal significance.

Nasal obstruction is a common complication of injury to the nose. Most nasal injuries will be followed by a period of temporary nasal obstruction either bilateral or unilateral, due to a temporary acute congestion of the nasal mucosa. If the injury, by virtue of fracture or displacement, causes narrowing of the airway in the nose, then nasal obstruction will be complained of. It is not necessary that a nasal fracture occur for an injury to the nose to produce nasal obstruction. The dorsum of the nose is most important in the preservation of nasal ventilation, and it is this

area that is most often deranged in nasal injuries. In order to obtain adequate relief from nasal obstruction, it may be necessary to do a rhinoplasty, rather than the conventional type of submucous resection, so often done in the past.

Anosmia and cerebrospinal rhinorrhea may follow nasal fractures involving the cribriform plate. The anosmia, besides being a disturbing symptom, will of course affect the efficiency and safety of workers who require a well developed sense of smell. The management of cerebrospinal rhinorrhea depends upon the extent of skull fracture. Occasionally a clear nasal discharge will persist, and the question of danger due to remote infection will have to be considered. After head injuries, one must differentiate quite often between the clear discharge of perennial vasomotor rhinitis and the clear discharge that occurs in cerebrospinal rhinorrhea. The latter condition is quite rare.

After a contusion or a fall or without fracture of the sinus walls, hemorrhage into the sinus is common. Generally this is of only passing significance. Occasionally a chronic antral infection may develop and require treatment. In the absence of infection, no treatment is required for hemorrhage of the antrum. Occasionally contusions in the region of the frontal sinus may activate a latent infection and eventually lead to the development of chronic fistulae of the anterior wall. Failure to recognize the underlying infected sinus as the cause of a chronic fistula in the area may lead to unnecessary prolonged treatment and disability. The cure is elimination of the infected sinus.

Acute and chronic infections of the sinuses are rarely encountered in ordinary industrial practice, but they may be seen in swimmers and may occur as a complication of industrial respiratory allergy, which is a common condition.

Perennial vasomotor rhinitis, with or without polyps and with or without asthma, is a common condition seen among workers in various industries. This will be discussed under the general heading of allergy.

Transient irritations and congestions of the nasal mucosa with the production of acute rhinitis due to the inhalation of irritating fumes is a recognized industrial condition, and ordinarily the problem is handled by provision of adequate ventilation. Many workers have to inhale fumes of acids, and in them perforations of the anterior cartilaginous septum are likely to develop. This condition is commonly seen in platers who work with chromic acid but may be seen in other industries where the fumes of acids are a necessary concomitant of their work. More frequently than not, these workers are unaware of the chronic nasal perforation. In any event, there is very little that can be done for them medically, except to prescribe bland emollients and measures to reduce the crusting that often occurs. From a prophylactic standpoint, of course, it would be most important to suggest that such industrial processes be carried out in an adequately ventilated environment.

Case 21.—B. W., a young woman, a factory worker, was struck on the nose by a falling box. X-rays revealed a comminuted fracture of the nasal bones. A few months after the injury she presented a substantial disfigurement of her nose and also complained of nasal obstruction. A rhinoplasty was performed, and the claim was finally terminated with a nominal award for facial disfigurement.

Case 22.—A. C., a steelworker, was struck on the right cheek by a piece of iron. X-rays showed a cloudiness of the right antrum. The patient lost only one day from his work; the contusion healed without incident, and x-rays taken several months after the accident showed clear sinuses.

CASE 23.—C. W., an elderly charwoman, sustained a contusion over the right eye. A persistent purulent exudate developed just beneath the supraorbital ridge. Finally x-rays were taken, and a chronic frontal sinusitis was demonstrated. When the sinus was treated surgically, the fistulous tract healed. Settlement was finally made on the basis of a facial disfigurement.

OCCUPATIONAL DISEASES AND INJURIES OF THE MOUTH AND LARYNX

Injuries to the teeth comprise the largest proportion of oral injuries. The employer is responsible for replacement of natural teeth and often even for repair or replacement of artificial teeth and dentures. Occasionally it is necessary to remove infected teeth as foci of infection to facilitate cure of a traumatic disease elsewhere in the body.

Fractures of the upper and lower jaws are also fairly common and present problems similar to those of the same condition in nonoccupational trauma.

Occupational disease of the mouth is rare. Recognition of the "lead line" in the gums may point to a diagnosis of lead poisoning. A Vincent's angina occurring in a laboratory worker was shown to arise from her work.

The larynx may be traumatized directly with resultant permanent hoarseness. More commonly traumatic hoarseness may develop in salespeople or singers. The finding of "singers' nodes" in a worker who must use his voice excessively confirms the diagnosis. It is sometimes necessary to advise a change in occupation.

The syndrome of globus hystericus occurs with some frequency in seamstresses, who have habitually kept a thread in their mouth and sometimes feel that it has lodged in their throats. They may continue to complain of a lump in the throat for long periods despite negative laryngoscopic studies.

CASE 24.—A. W., a laborer, was struck on the face by a beam. Two incisor teeth were knocked out, and the left interior ramus of the mandible was fractured. The lower jaw was cured and ultimately healed. A dentist restored the loosened and missing teeth. After three months the patient resumed work, and the claim was finally settled with a finding of a facial disfigurement.

OCCUPATIONAL DISEASE OF THE LUNGS AND BRONCHI

The lungs and bronchial tubes are quite susceptible to harmful effects of noxious fumes, harmful excessive mineral and nonmineral dusts, and various sensitizing materials.

Tuberculosis in hospital personnel also occurs frequently. The otolaryngologist plays an important role in the diagnosis and management of such. Laryngoscopic and bronchoscopic studies are frequently indicated to determine the extent of the pathology and also to rule out unrelated pathologies.

The role of sinusitis in the development of a chest complaint must frequently be evaluated. For successful treatment of many lung disorders, sinus pathology must be eradicated or treated.

Silicosis and associated dust diseases are generally seen and handled by chest physicians. Similarly cases of tuberculosis for the most part are treated by tuberculosis specialists and the ear, nose and throat specialist is called in only occasionally for evaluation of treatment of laryngeal lesions or for bronchoscopic evaluation of pathology.

The allergic and nonmineral dust diseases are frequently seen by ear, nose, and throat specialists, and it is patients suffering from these diseases for whom most can be done.

CASE 25.—C. C., a salesman for 26 years, complained of recurrent hoarseness of several years' duration. The ear, nose, and throat examination disclosed nothing significant except several small "singers' nodes" on the vocal cords. The nodes were removed, but hoarseness recurred and ultimately the patient had to change his occupation. The medical treatment and disability were held to be compensable.

INDUSTRIAL ALLERGY OF THE RESPIRATORY TRACT

Any competent rhinologist, not restricted in his outlook or fascinated by the curvatures of the nasal septum, soon realizes that allergies of the nasal respiratory mucosa play a major role in the production of nasal respiratory symptoms of the patients who come to him for relief. Failure to recognize such factors has led to much criticism and general popular dissatisfaction with the rhinological practice of the past several decades. In industry, allergy of the respiratory tract is an important problem. It causes much distress and disability and is responsible for extensive medical liability.

Workers in the fur industry are particularly susceptible to allergic disease of the respiratory tract. The aniline dyes used in this industry have been shown to be potent sensitizers and have caused a considerable amount of respiratory disease in the fur and allied industries. Most often it is the actual dyeing of furs which is most hazardous, but in other workers such as finishers, stretchers, or sales people symptoms and disability may occasionally develop. The length of employment does not appear to be of much importance in the development of symptoms. Occasionally changing of the formula of the dye used will precipitate allergic symptoms. In these workers there may develop all types of respiratory disorders varying from a simple perennial vasomotor rhinitis, with rhinorrhea and alternating nasal obstruction as a presenting symptom, to full-blown nasal polyps and even with complicating chronic sinusitis. They may also acquire extensive and disabling types of bronchial asthma with emphysema or bronchiectasis or chronic bronchitis as complications.

Bakers may occasionally show sensitivity to molds and acquire allergy of the respiratory tract. Baker's asthma is a well known and old industrial disease. Millers and handlers of cereals also come within this category.

Gardeners may show sensitivity to flowers or grasses. It is a clinical observation that sudden massive exposure to pollens may precipitate a latent hay fever.

In other industries where various types of dusts are present in an inordinate degree, workers will complain of nasal conditions and attribute these to their occupations. Inasmuch as in New York State particularly, a broad interpretation of the compensation law indicates that any and all conditions that can be shown to be caused by occupation come within the scope of the law, it is an important problem to differentiate between respiratory disease actually due to the industrial environment and respiratory disease occurring as an incident of any environment and based upon individual susceptibility.

The diagnosis of industrial allergy of the respiratory tract is based upon the history of exposure to substances which are known to have produced respiratory allergy in the past, in an industry in which symptoms and disability definitely develop in a large proportion of workers. Physical and clinical examinations, supplemented by skin tests with selected antigens and perhaps by inhalation tests of substances suspected to be guilty of causing the allergy, are important and invaluable adjuncts in diagnosis. The therapeutic tests of cessation of employment followed by improve-

ment in symptomatology and resumption of employment followed by recurrence of symptoms will often be more effective than other diagnostic procedures.

The best treatment of severe industrial allergy of the respiratory tract obviously is a complete change of occupation. The economic stakes are too large to warrant such change routinely. Occasionally desensitization and local supportive measures may enable a worker to continue at his chosen trade. Removal of nasal polyps, when they are present, and treatment of chronic sinusitis as part of the therapeutic regimen are often indicated. In severe cases, management may be necessary as long as the patient lives.

CASE 26.—A. G. has been a furrier for 16 years. For the past few years he has had recurrent "colds" with nasal obstruction. A few weeks before the examination he had several asthmatic attacks. The examination revealed nasal polyps bilaterally, and moist musical rales over the lung fields. Skin testing showed positive reactions to dust and to paraphenylenediamine. An inhalation test with a 1: 1,000 solution of the paraphenylenediamine dye produced an asthmatic attack. The patient stopped working. His polyps were removed and his sinusitis treated. He was also given desensitization and symptomatic treatment for his asthma. He made several attempts to resume work, but each time there was exacerbation of the asthma and nasal symptoms. He finally had to change his occupation. Despite this, his symptoms recurred occasionally, and it has been necessary to keep him under treatment to date.

SUMMARY

There are many diseases and injuries covered by workmen's compensation laws that come within the province of otolaryngology.

Contact dermatitis of the auricle, frost bite, welding burns of the external canal and tympanic membrane, direct injuries to the drum, otitis media, and aggravation of the otitis media occur quite frequently.

Head injuries often cause changes in the cochlea and labyrinth, and excessive industrial noise causes widespread hearing loss.

Facial paralysis is often compensable and presents problems of concern to the otolaryngologist.

Rhinoplasty and plastic surgery have enhanced the management of nasal and facial injuries by the ear, nose, and throat specialist.

Industrial allergy of the nasorespiratory tract is a large field in which the otolaryngologist can perform useful service.

35-02 168th St. (58).

CORTICOTROPIN (ACTH) AND FENESTRATION

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NOW THAT a preliminary report on the possible value of corticotropin (ACTH) for the fenestration operation has appeared[1] and another is in print,[2] additional comment seems warranted.

Although in nearly every other field of medicine the literature is flooded with reports on corticotropin, hardly any comment is given in the field of ear, nose, and throat research.[3] Only recently Hollander, commenting on this hormone,[4] stated, "Probably in the field of ear, nose and throat surgery there is a relative lack of conditions where these hormones are really vitally needed." The indications for its use that Hollander gave were the well-known allergic diseases. Now for a proper understanding of the role corticotropin can play in any particular case, the indications for and the dangers of its use, we should be thoroughly acquainted with the biological principles that Selye[5] has pointed out. The interested reader is referred to the original literature for a full account of the many contributions that corticotropin and cortisone have made to our arsenal.

To approach the problem of fenestration surgery, I will quote from a lecture that Selye gave in Holland in the summer of 1950[6]: "The gluco-corticoids generally inhibit excessive proliferation of fibrous tissue, the formation of intercellular protein deposits (e. g., hyalinosis, collagen disease, allergies) and excessive granulomatous defence reactions against those local irritants which stimulate fibroplastic inflammatory reactions in mesenchymal tissue." This effect is beautifully illustrated in the experiments made by Ragan.[7]

It occurred to me while reading Selye's paper that the very fact that corticotropin has an inhibiting effect on wound healing might be of paramount importance in fenestration surgery. In my opinion, it is not the osteogenesis, the bony closure of the fenestra, that forms the major problem in fenestration, since the newer

1. Angeluscheff, Z.: Otosclerosis and ACTH, A. M. A. Arch. Otolaryng. **54**:200 (Aug.) 1951.

2. Lansberg, M. P.: De betekenis van ACTH voor de Otologie, to be published.

3. Williams, H. L., and Hochfilzer, J. J.: Effect of Cortisone on Idiopathic Granuloma of the Midline Tissues of the Face, Ann. Otol. Rhin. & Laryng. **59**:518-531 (June) 1950.

4. Hollander, J. L.: Practical Problems in the Use of Cortisone and ACTH, Laryngoscope **61**:565-572 (June) 1951.

5. Selye, H.: The Physiology and Pathology of Exposure to Stress, Montreal, Acta Inc., 1950; Textbook of Endocrinology, Montreal, Acta Endocrinologica, Université de Montréal, 1948, p. 797.

6. Selye, H.: Het Hormoon, Oss, Netherlands, 1950, p. 37.

7. Ragan, C., and others: Effect of Cortisone on Production of Granulation Tissue in the Rabbit, Proc. Soc. Exper. Biol. & Med. **72**:718-721 (Dec.) 1949.

techniques will eliminate this danger to a major extent, but it is the very first wound reaction, the serous labyrinthitis, that threatens success of the results or, to quote from a survey by Shambaugh[8]: ". . . serous labyrinthitis is a major cause for failure. The problem of what causes this condition and how to prevent it is still far from solution, but on its solution largely depends further improvements in results with the fenestration operation."

The many factors that play a role in wound reaction are extensively described by Menkin.[9] These investigations show that certainly something more complicated than a little extra histamine is involved, as was suggested in earlier works. In order to annihilate the often deleterious effect of the serous labyrinthitis that to a major or minor extent will leave the fenestration patient with a perceptive loss, we must do more than just give one antihistamine or another, and if one of the effects of cortisone is to suppress this very wound reaction, it seems logical to take advantage of this property and try it.

In order to substantiate my theories, I felt it wise to get experimental evidence, and for this reason I planned histological experiments in October, 1950. The skull of the rabbit was selected for this purpose, since in it one deals with a situation more or less comparable to labyrinthine surgery: periosteum, bone, periosteum, and cerebrospinal fluid.

The results on the first series of rabbits confirmed my expectations, showing an inhibited wound reaction in the corticotropin-treated animals. The second series was begun to confirm the first results, but at the same time, I felt that a more pertinent experiment had to be sought in order to draw conclusions and make predictions as to the effect of corticotropin in otosclerosis surgery. What had to be proved was that corticotropin or cortisone had a beneficial effect on the postoperative hearing result in otosclerotics. Now this result is largely determined by the following factors: (1) preoperative cochlear function; (2) postoperative impedance relations of the cavity and the exact place of the fenestra (this point may become of surgical importance when more is known about the accoustic properties of the cavity and the optimal phase relations between the two fenestras [von Békésy]); (3) scrupulous surgical technique as extensively described in the pertinent literature; (4) wound reactions—(*a*) serous labyrinthitis, (*b*) fibrous thickening of the flap, and (*c*) bony closure of the fenestra.

As stated above, it is the serous labyrinthitis that endangers the perceptive qualities of the ear and thus must be considered the most distressing effect. Secondly, being the primary wound reaction, serous labyrinthitis probably creates the conditions that ultimately bring about the two later wound effects.

Whereas theoretically corticotropin or cortisone was all I needed to reach this goal, I thought, however, that functional evidence in the animal experiment seemed desirable in order to get an idea of dosage and possible undesired effects. Regarding the latter, I feared the following developments: (1) malnutrition of the flap and possibly necrosis; (2) hemorrhages from the flap into the labyrinthine cavity; (3) no, or delayed, fixation of the flap on the fenestral rims; (4) rebound phenomenon after the medication was stopped.

8. Addin, L. E., Jr., and Shambaugh, G. E.: A Study of Long Term Hearing Results in Fenestration Surgery, A. M. A. Arch. Otolaryng. **53**:243-256 (March) 1951.

9. Menkin, V.: Modern Concepts of Inflammation, Science **105**:538-540 (May 23) 1947.

I know that a high concentration of the cortisone must be administered to obtain the desired catabolic effect.[10] For that reason cortisone by local application seemed superior to corticotropin. I think that a slow-absorbable cortisone ointment to fill the cavity will be the best way to apply this hormone. Another possibility is daily injections of cortisone suspension into the cavity. A further advantage of cortisone topically administered over corticotropin is that it circumvents the many systemic side-effects of this potent hormonal substance.

Clinical proof of the necessity of high dosages was obtained when I operated on two patients on April 1, 1951, and gave daily corticotropin injections for six days starting with six times 8 mg. and gradually lowering the amount to six times 3 mg. Although good, the result was far from spectacular.

I am not very much afraid of the mentioned rebound phenomenon; the "self-limiting character" of the wound reaction will guarantee absence of dangers from this angle. It would take too long to explain here on theoretical grounds the biologic working mechanism of corticotropin and cortisone, but the fenestration surgeon should understand that making a wound in the most concealed part of the human skull is a most unbecoming trauma to the minute structures of the inner ear. Nature's response to our brutal interference must be respected as being one of preventing further damage. This is accomplished by anti-infectious and antitraumatic barrier building as anywhere else in the body. Now that because of sterile surgical technique and the aid of antibiotics infection is no longer feared, the wound reaction has lost its life-saving quality; what we now desire is to prevent barrier building, and therefore nature should be given the tools to produce a clean wound.

Then why was it so hard to get pertinent animal experiments to confirm my theories? The answer is manifold:

1. One does not have otosclerotic animals at one's disposal. Artificial fixation of the stapes with dental cement would make the technical problem much greater, and many new factors would be brought in. I therefore decided to drop this idea and make comparisons between corticotropin- or cortisone-pretreated animals and controls after traumatizing the labyrinth. If no serous labyrinthitis evolved in the former category, the results would certainly give welcome information.

2. What animal should be selected? The corticotropin effect differs considerably in different animals and in different aspects. The guinea pig, with its many desirable qualities for inner ear experiment, was the first to be considered. Unfortunately, several reports make it seem probable that the wound reaction in the guinea pig is not influenced by corticotropin.[11] Reports with a contrary conclusion, however, have also been published.[12]

3. What operation should be performed? To imitate a fenestration seems worthless, since there is no stapes fixation in the animal. Furthermore, the postoperative result, as outlined above, depends on several factors. Thirdly, it is very hard, if not impossible, to do a fenestration with building of the flap, etc., in most animal species except the monkey. Therefore, I decided that all that was needed was just a sterile labyrinthitis extending to the cochlea, and I felt that a small incision in

10. Selye, H.: Personal communication to the author.

11. Brit. J. Exper. Path. (May) 1951.

12. Cameron, G. R.: Tissue Reactions, Lancet **1**:1212 (June 2) 1951.

the upper cochlear turn would be all that was needed to get a small hemorrhage in the labyrinth. Of course, this is far from ideal.

4. What testing procedure should be followed? Apart from the histological evidence that was desired, I wanted a direct answer to the question: What does the animal hear? Microphonics give valuable information. It must be expected that the labyrinthitis will have its effect on hearing. From several experiments [13] I know, however, that hearing loss, even if of a purely end-organ origin, as proved histologically, does not correspond in a simple way with changes in the microphonics. Conditioned or unconditioned response is for that reason preferable. This implies, however, the necessity of deafening the untested ear, one more burden to our technical problems. The pinna reflex in the guinea pig in our experiments proved to be appropriate for this purpose. However, if the guinea pig is, for the reasons given earlier, not a suitable animal, I must turn to another test.

Many more problems were encountered, and to tell the story of trial and error would fill many more pages. It is my opinion now that the possible advantages of cortisone treatment are so paramount that a clinical trial should no longer wait for experimental evidence that will bring so many difficulties with it in order to give pertinent information. I am fully aware of the fact that every fenestration operation is a risk as to the hearing result.

When so much can possibly be gained for so many by a principle that is now available, should it be withheld from the patient any longer because we do not have final proof of its beneficial effect from animal experiments?

SUMMARY

A description is given of the effects of corticotropin (ACTH) and cortisone on wound healing, and its relations to the fenestration operation are discussed. Many technical problems involved with the animal experiments are outlined.

ADDENDUM

While I was writing this manuscript, the *Laryngoscope*, issue of August, 1951, arrived. On page 707, Hilger mentions the possible role corticotropin might play in curing labyrinthine crises of Ménière or pseudo-Ménière character. Since this idea has had our ample consideration concurrently with the fenestration problem for many months, I hope to deal with this aspect of corticotropin therapy in a following article in which several other ear, nose, and throat indications for the use of corticotropin will be discussed.

Scientific and technical aid was given by Professor Querido, Professor Duff, and their staff and several other Dutch colleagues. N. V. Organon provided the corticotropin. A grant was supplied by Hippocrates Studiefonds.

13. Schuknecht, H. F., and others: An Experimental Study, Ann. Otol. Rhin. & Laryng. **60**:273-290 (June) 1951.

TREATMENT OF OROANTRAL FISTULA

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IT IS THE intention of this article to discuss the problems in the treatment of oroantral fistulas and to re-present an operation for their closure that has not received the recognition it deserves.

Alveolar fistulas may be divided into two types: the acute, or those of recent origin; and the chronic, or those of long duration.

ACUTE OROANTRAL FISTULA

Extraction of one of the upper molar teeth is the usual cause of acute oroantral fistulas. They are therefore first seen by the dentist, although the rhinologist may be consulted to help care for the sinus complication. Rhinologists are fairly well agreed regarding the management of the acute condition, as mentioned in Barton's [1a] summary. The fundamentals of treatment of acute oroantral fistulas include (1) adequate antimicrobial therapy, preferably penicillin; (2) no manipulation through the fistula (which includes no probing, no packing into the antrum, and no irrigation of the antrum through the alveolar opening; if seen early enough, the gingival margins should be sutured by the dentist); (3) care in blowing the nose, so as not to force air through the fistula; (4) intranasal diagnostic irrigation if symptoms suggest an infection of the antrum. If pus is found, subsequent irrigations should also be carried out by the nasal route. When the maxillary sinusitis fails to respond to antibiotics and a reasonable number of irrigations, a surgical window should be made in the inferior meatus to facilitate aeration and drainage.

After the relief of the sinus suppuration, and with no instrumentation of the fistula, most acute oroantral openings will close spontaneously.

Unfortunately, in acute cases patients are not commonly seen by the rhinologist, in spite of the frequent pleas for teamwork with the dentist. One reason for this

Read before the Chicago Laryngological and Otological Society, Feb. 4, 1952, with the presentation of a motion picture, "An Operation for the Closure of Oroantral Fistulas."

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1. (*a*) Barton, R. T.: Modern Management of Oroantral Fistula, Arch. Otolaryng. **50**:732-739 (Dec.) 1949. (*b*) Ashley, R. E.: A Method of Closing Antro-Alveolar Fistulae, Ann. Otol. Rhin. & Laryng. **48**:632-642 (Sept.) 1939. (*c*) Hill, F. T.: Management of the Alveolar Fistula, Arch. Otolaryng. **40**:167-170 (Sept.) 1944. (*d*) McQuiston, R. J.: Maxillary Sinusitis of Dental Origin and the Management of Antral Fistula, Ann. Otol. Rhin. & Laryng. **54**:373-383 (June) 1945. (*e*) Hersh, J. H.: Plastic Repair of a Large Alveolar-Antral Fistula, Arch. Otolaryng. **43**:141-142 (Feb.) 1946.

reluctance to consult with the rhinologist may be that the dentist feels some embarrassment at having "broken into the antrum." It seems hardly necessary to stress the close relationship of the dental apices to the floor of the antrum. With an apical abscess the thin floor of the sinus may become necrotic and be unavoidably damaged when the tooth is removed. Opening into the antrum in this situation must not be considered to reflect on the skill of the dental surgeon. He may be criticized for his care of the patient when this complication occurs if he packs into the antrum or irrigates it frequently through the tract. These procedures promote epithelization and lead to a chronic fistula.

CHRONIC OROANTRAL FISTULA

After six months a fistula may be considered as chronic. The pathological condition consists of an epithelized tract through the alveolus into the antrum, a chronic infection of the maxillary sinus, usually with anaerobic organisms, and some localized osteitis about the margins of the bony opening. There is little possibility of a chronic oroantral fistula healing spontaneously. Abatement of the suppuration in the maxillary sinus is not likely to be followed by closure of the alveolar opening. Reinfection of the antrum is common as long as there is a communication with the mouth.

Rhinologists [1] writing on this problem are in agreement that the primary requisite for closure is cure of the sinus infection combined with some type of plastic operation. It is often recommended that this be done in two stages. The sinus surgery is done first, and after the infection has subsided the fistula is closed.

Dental surgeons [2] also mention treatment of the ever-present sinusitis. Most of them do not emphasize that successful closure is dependent on previous relief of the sinus infection. Oringer,[2d] a dentist, was an exception. He stated that no type of plastic closure of the fistula can succeed unless the antrum is in a healthy state, free of all infection.

A simple intranasal window will suffice to end the antral suppuration in most instances. It is probably aeration rather than facilitated drainage which makes the window operation remarkably effective in anaerobic infections of the sinus. The Caldwell-Luc operation has the great advantage of permitting inspection of the interior of the antrum for the discovery and removal of the frequently present foreign body. A tooth is most commonly found, also gauze packing, gum, food, and sequestrae from the antral floor. The foreign body may not be visible in roentgenograms.

A number of plastic operations for closure of the fistula have been described.[4] The commonest operation favored by both oral surgeons and rhinologists consists of raising and fashioning a flap from the hard palate, rotating it over the bony

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2. (*a*) Berger, A.: Oroantral Openings and Their Surgical Correction, Arch. Otolaryng. **30**:400-410 (Sept.) 1939; (*b*) Principles and Technique of Oral Surgery, Brooklyn, Dental Items of Interest Publishing Company, 1925, pp. 191-202. (*c*) Ferber, E. W.; Gwinn, C. D., and Grimm, D. H.: Operation for the Repair of Oroantral Fistulae, J Oral Surg. **6**:139-146 (Jan.) 1948. (*d*) Oringer, M. J.: Plastic Closure of Alveolar Antral Traumatic Perforations and Chronic Fistulae, Presenting an Improved Technique, Oral Surg. **3**:410-420 (April) 1950.

3. Footnote deleted.

4. (*a*) Footnote 1*b*, *c*, *d*, and *e*. (*b*) Footnote 2. (*c*) Proctor, B.: The Closure of Oromaxillary Fistula: A Preliminary Report, Laryngoscope **56**:46-47 (Feb.) 1946.

defect, and suturing it in place. Proctor [4c] described a method of plugging the opening in the alveolus with a cone-shaped piece of costal cartilage and then covering it with a flap. His was a preliminary report.

In 1939 Berger,[2a] a dentist, outlined his method of forming a flap from the lateral aspect of the alveolus. He had described a combined palatal and alveolar flap earlier in his book.[2b] Others mention obtaining the flap from the alveolar process. The unique feature of Berger's operation is the incision of the periosteum at the base of the flap. This allows free mobilization by utilizing the loose buccoalveolar fold. The flap may be pulled down "like a window shade" with as much tissue as is necessary to sew well beyond the margins of the bony defect. The amount of tissue that may be gained by this maneuver must be seen to be appreciated. The surface of the flap presented to the antrum is lined with periosteum. It thus does not meet the requirement of correct plastic surgery that repair of the wall of a body cavity must have an epithelial lining as well as covering. Double surface flaps have been described.[5] They would seem quite involved for the occasional operator, not accustomed to plastic surgery techniques. The fact that the periosteal flap does succeed is probably due to the comparatively small size of the bony opening, and the fact that healthy antral mucosa will soon cover the periosteal bridge.

The following case is illustrative.

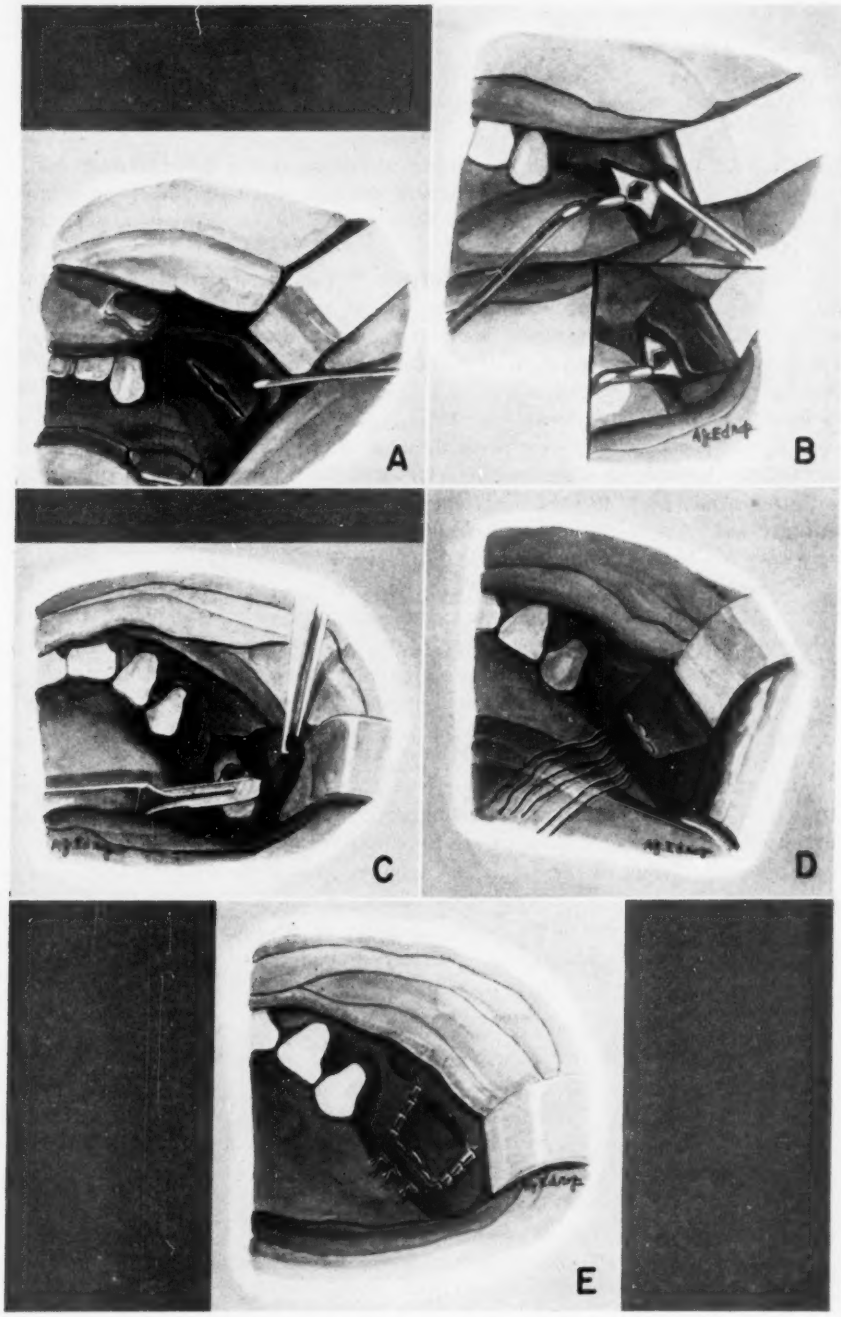
REPORT OF A CASE

A 25-year-old man had had a left second upper molar tooth extracted 10 years before. Six months prior to the time he was observed, a dental roentgenogram disclosed the presence of a remaining tooth root. Removal of the root was attempted, but it was apparently lost in the antrum. Following this there was profuse foul discharge from the left nostril, pain in the left cheek, and the escape of air about the gum on blowing the nose. These complaints were still present on admission.

On examination there was profuse purulent discharge observed in the left middle meatus. The left antrum was dark on transillumination. A roentgenogram also disclosed marked clouding of the left antrum but no evidence of a foreign body. Irrigation of the left maxillary sinus yielded a quantity of thin, foul green pus. There was a small fistulous opening into the antrum from the region of the second molar tooth. A left Caldwell-Luc operation was performed under local anesthesia. The sinus membrane was markedly diseased. Buried in the thickened lining was the segment of tooth. The entire lining membrane was removed, and a large window made under the inferior turbinate. There was prompt cessation of the sinus discharge and irrigations were clear within a few days.

Technique of the Berger Operation.—Five weeks later, the patient was readmitted for closure of the fistula. This was carried out by the method described by Berger.[2a] Anesthesia was obtained by local infiltration with 1% procaine hydrochloride solution. The incision was made (Fig. *A*) horizontal to the alveolus through the fistula down to the bone. Elevation of the periosteum disclosed the size of the bony opening (Fig. *B*). (It is always found much larger than the mucosal fistula.) After the location and size of the bony defect were determined, the two vertical incisions to form the flap were made (Fig. *B*), fashioning it considerably larger that the opening to be covered. The epithelial lining of the bony fistula was removed, with all granulations and diseased bone. The flap was then turned outward, and the periosteum incised horizontally (Fig. *C*) at the base. This allowed the flap to be pulled down without tension, applied over the bony opening, and secured with mattress sutures (Fig. *D*). The medial edge of the flap was placed well beyond the border of the fistula in the bone. Single sutures were used on the rest of the margins of the flap (Fig. *E*). Penicillin was administered for three days. The flap healed by primary union. The sutures were removed and the patient was discharged in one

5. Footnote 1*b*, *c*, and *e*.



Technique of the Berger operation (slightly modified). *A*, a horizontal incision is made parallel to the alveolus, through the fistula, to bone. *B*, elevation of the periosteum of the two flaps reveals the location and size of the bony opening. The epithelial lining is removed, with granulations and any softened bone. Insert shows two divergent incisions made through the periosteum, extending upward to the buccoalveolar fold. The flap is fashioned considerably larger than the opening to be covered. The scar tissue of the medial edge of the incision is removed, so that the suture line will be well medial to the edge of the bony fistula. *C*, the flap is turned outward, and the periosteum incised horizontally at the base. This allows free mobilization by utilizing the buccoalveolar fold. The periosteum only must be cut so as not to sever the blood supply of the flap. *D*, mattress sutures are used to secure the flap over the bony fistula. *E*, the remaining edges are united with single sutures.

week. Examination one month later revealed the fistula healed. Irrigation of the sinus gave a clear return.

Following this operation there is some alteration of the buccoalveolar fold, resulting in a less prominent angle. The fold tends to deepen gradually in a few months. There has been no problem in having a prosthesis fitted in any of the patients in this series.

I have performed the Berger operation on 10 patients, with only one failure. In that case there was no pus on irrigation of the antrum, and the infection seemed quiescent. A window was made at the same time as the plastic closure. There was a prompt exacerbation of the sinus infection, with subsequent purulent drainage about the flap and failure of union. The flare-up of the sinusitis was thought to be due to the closing off of the fistula and the fact that postoperative swelling did not allow free ventilation through the window opening. The antrum was sealed off, setting up ideal conditions for anaerobic organisms. The nine patients with successful results had the sinus operation performed some five or six weeks before plastic closure was done. Penicillin administered to some of the patients may have contributed to the favorable results.

The success obtained with Berger's operation would seem to recommend wider employment of this method. In most of these cases one or more previous attempts at closure had been made by other procedures. The operation is readily performed. It seems especially adapted for fistulas on the lateral aspect of the alveolus (a common site) or where the opening is located between two remaining teeth.

SUMMARY AND CONCLUSIONS

The treatment of acute and chronic oroantral openings is discussed. Administration of penicillin, avoidance of manipulation of the opening, and intranasal drainage of the associated sinusitis are recommended in the case of recent fistulas.

Successful closure of chronic fistulas requires cure of the sinus suppuration by antrotomy, followed in a few weeks by a plastic operation.

A description is given of the Berger operation of raising a flap from the lateral aspect of the alveolus. Wider use of this method is advocated.

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RADIOGRAPHY OF THE SUPRAGLOTTIC SPEECH ORGANS

A Survey

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WILHELM KONRAD VON RÖNTGEN published his epochal paper, *Eine neue Art von Strahlen,* in December, 1895. Within a few months the knowledge of his rays had spread with incredible rapidity; many investigators started application of them to their special realms.

The apostle for rhinolaryngology in general, and for phonetics in particular, was Max Scheier in Berlin. The interval of only a year and a half from the discovery of the rays was needed by Scheier before he announced, on June 10, 1897, a lecture at a congress for internal medicine in Frankfurt am Main entitled, "The Application of X-Ray in the Physiology of the Voice and the Speech." This paper probably must be considered as the first on the topic in question.

Scheier's presentation reflected by no means a "fleeting purpose," as he followed it in the same and the next year by papers given in Brunswick and in Berlin, and by publications for the general medical public, in *Deutsche medizinische Wochenschrift* and in *Allgemeines medizinisches Zentralblatt*; for the rhinolaryngologist in *Archiv für Laryngologie* and *Archives internationales de laryngologie, d'otologie et de rhinologie;* for the philologist-phonetician, in a supplement to the periodical *"Die neueren Sprachen."* In 1899, he discussed the question in a monograph in Drasche's collection. After an intermission of 10 years, Scheier returned to this field during 1908 and 1909 with several lectures and publications.

Roentgen's method was so clear-cut, on the one hand, and the needs and shortcomings of the investigations into the production of the speech sounds were so obvious, that by perusing these pioneering papers of Scheier it is possible to foretell the entire later development of this specialized technique. The present survey is interested in the development of x-ray visualization of the position and the movements of the anatomical elements that compose the organs of the resonating tube. It is restricted entirely to the technical aspect of the problem.

By placing the face of the subject, parallel to its midline, against the x-ray screen, Scheier saw for the first time "an ideal cross section of the organs of the head and the neck." Starting immediately after the publication of Roentgen's paper, Scheier was able, by using better tubes, to avail himself of improved technique

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even within the first year. He could see no advantage in photography, but put a thin film of celluloid on the screen, covered it with paper, and traced the contours with pencil.

In June, 1897, he claimed already to have made a thorough study of the production of the consonants and stated that the first and second articulation points were the easiest to examine, these two being (1) between the lower lip and the upper lip, or the upper incisors, and (2) between the tip of the tongue and the teeth, or the hard palate. At this early date he had already considered phonetical implications and discussed the new method in critical comparison with previous purely observational and experimental procedures.

Two points of importance were cleared up in the course of Scheier's introductory work. First, in order to have an undisturbed picture of the organs participating in the production of the speech sounds, he used edentulous subjects. He thereby initiated a discussion by no means ended even today: Is a mouth with as few teeth as possible preferable, or should one, rather, try to work with a complete dental arch and apply artificial means to outline the organs in question? Second, since the time of Merkel it had been known that each speech sound has a certain physiological latitude —that within this boundary the organs move freely without impinging on the sphere of another sound, as hearing would betray. Scheier showed that limits, though by no means rigid ones, can be drawn for the articulations that may be considered normal.

In 1898, Scheier made studies of a theatrical imitator of instruments, to determine, with the help of the newly discovered rays, the necessary configuration of the resonating tube when the different sounds are produced. The mechanisms of swallowing, respiration, snoring, ventriloquism, and others were examined. In these experiments he included the study of the movements of the diaphragm, and so became the first investigator of the entire speech mechanism from the diaphragm to the lips so far as it can be followed by roentgenography.

Ten years later Scheier was able to report reduction of the exposure time, originally 10 to 15 minutes, to 25 to 50 seconds; later, with the help of the "Grissonator," he reduced the exposure time to half a second. He saw clearly that by achieving an exposure time of fractions of a second, it would be possible to prepare cinematographic documents of the speech mechanism. He emphasized that on the screen it is much easier to observe motion than a resting position. He later gave up painting the dorsum of the tongue with barium or marking it with small pieces of "stanniol," as it was found that any foreign body in the mouth influences the pronunciation of vowels.

Scheier, in his activity throughout 10 years, clarified practically every major problem of this method of investigation, so far as the technique of his time permitted. He also sketched clearly its further possibilities, should a higher technical level be reached in the future; so when speaking of cinematography, he called its advent only a question of time. On the other hand, confronting it with other techniques of experimental phonetics, he assigned the method its proper place, explaining that it should always be used not to replace but to supplement them.

In an early period, Lloyd published (1897-1898), in his studies on structure and causation of the speech sounds, profile charts of the "ordinary articulations" of English vowels. These charts were reproduced in the "Elements of Phonetics," by

Rippmann. Neither of them mentioned anything regarding technique or, indeed, whether these profile pictures were obtained by roentgenogram at all.

In a long series of investigations, Gutzmann and Mendelsohn (1899) already clearly saw the velum, its outlines and movements during swallowing and speech.

Jespersen (1904) wanted to replace the term "experimental phonetics" by "instrumental phonetics," "objective phonetics," or "machine phonetics." In collaboration with Mygge "soon after Roentgen," he tried the new technique but gave it up when he did not find the pictures clear enough. Speaking of Scheier, he expressed doubts regarding Scheier's results, stating that they might have been based on self-deception!

Barth (1907) secured the collaboration of a competent roentgenologist, Grunmach. Thus, as had been done by Jespersen and Mygge, a working team was formed. This scheme was repeated by others in the following periods under review, with excellent results. Head and occiput of the subject were supported and fixed in their position. The exposure time was 12 seconds. The most prominent of their subjects was a "Kammersänger" able to build the vowels with an unsurpassed clearness and impeccable resonance. Contour-marking was done according to the following technique: a thin, light, metal chain ending in a small ball was placed on the median line of the tongue. The ball was in the vallecula close to the middle glossoepiglottic ligament, with the free end of the chain hanging from the mouth. A similar chain was used to mark the position of the soft palate; it was introduced with the help of Bellocq's cannula through the floor of the nasal cavity in such a way that the ball came to lie just above the free edge of the soft palate. No cocaine was used. In a person of normal sensibility no reflex movements were elicited. Pronunciation and clearness of the vowels was not at all influenced by this contraption.

Barth and Grunmach emphasized, as had been done already by Scheier, that, regarding behavior of the resonating tube, x-ray investigation found itself in perfect harmony with results secured by leading physiologists in the pre-Roentgen era.

Meyer (1907) mentioned that he had experimented in 1905 with a method similar to that of Barth and Grunmach, but his results were published only in the same year as the communication of Barth and Grunmach, 1907. Meyer intensified the profile outlines of the tongue and pharynx by a chain of small lead plates connected by two silk strings; he used on the palate the same kind of plates, pasted on adhesive. The weight of one plate was about 0.05 gm. and of the whole chain about 0.5 gm. The pressure was too insignificant to be felt by the tongue at all. With the central ray directed against the second upper molar, and with an exposure of one minute, the sound was repeated eight times in the course of this experiment; the last detail was considered an advantage of the method, as it made it possible to obtain a cross section from eight slightly different pictures.

By this method of repeating each utterance eight times, Meyer found later (1916) that the latitude of variations for each articulation position is not as great as had been imagined previously. (Merkel, mentioned above in connection with the investigations of Scheier.)

Fröschels, in collaboration with the roentgenologist Haudek (1911), obtained excellent pictures in spite of the circumstance that one of their subjects, who provided the greatest number of pictures, had a complete denture. The subject even had several gold fillings, and the tip of the tongue was frequently obscured by these.

With a thin wooden applicator, paste of bismuth (carbonic bismuth with sugar and water) was painted as a fine line on the lips, the tongue, the inferior aspect of the soft palate, and the midline of the posterior wall of the pharynx; the last two regions had been cocainized beforehand. This painting had to be renewed after about two pictures. The articulation started before and was terminated after the exposure, the latter lasting not longer than four or five seconds. For some pictures two bismuth lines on the tongue were used: one run in the midline, the other on the edge. The prints were retouched exactly according to the plates, as prints taken directly from the plates did not turn out sufficiently sharp. Fröschels and Haudek confirmed, too, a surprising accord between their photographs and the results that Bruecke and Merkel had secured by auto-observation.

Eijkman (1914) experimented with himself as the subject. The head was supported at the back, the plate placed as close as possible to the right jaw. Comparison of the various photographs was facilitated by a rectangular cross of copper wire on the frame. The roentgen tube was suspended a little behind the angle of the jaw and slightly above the level of the slot of the mouth, so that it was not the median line of the surface of the tongue that was shown in the photographs but one slightly to the right of it. The exposure time was about 1¼ seconds; some bismuth powder was strewn on the tongue. Tracings were prepared from the photos by means of carbon paper.

Schilling (1914), using an exposure of 1/100 of a second, rejected fixation of the head, in order to avoid any influencing of the singer. The singer, however, was asked not to change the position of his head between two exposures. Schilling conceded that errors are possible with this method and so did not put the main emphasis on measurements.

Navarro (1916), using an exposure of 3/10 of a second, likewise discarded metal plates or castings. He prepared tracings of the negatives, though this was done with great difficulty as regards the tongue; the velum was left out entirely because it could not be seen clearly enough. The clearest outline was furnished, as was to be expected, by the hard palate and the mandible.

Panconcelli-Calzia (1919) has shown that roentgen-polygrams, as advocated by Levy-Dorn and Silberberg for gastral movements, can be adapted to the study of the production of vowels and consonants in the larynx and the resonating tube. The moving organs were photographed several times on the same plate, in short intervals. The technique was about the same as the one used in phonetic x-ray photography in general. Measurements of a similar "multiplum," or group of superimposed pictures, was possible with the help of markers. A formula could be developed, characteristic of the particular movement. Stereophotography can be added to perfect the method.

Wiebe (1921) discussed the measurement of roentgen-polygrams. True, this measurement is not necessarily exact and complete. Even so, it is of paramount importance, approximative numerical data being more valuable than any vague description. He explained the application of the axial cross, necessary when working with polygrams with two or more sounds produced and fixed on the same plate.

Barclay and Nelson (1922), whose manuscript was stolen and could be published afterward only in a very abbreviated form, found after a number of experi-

ments that the best practice is to make a paste of bismuth carbonate with petrolatum and apply a line down the middle of the tongue and frenum, with a corresponding line on the soft palate. As a further aid a fine bismuth powder was insufflated on the back of the tongue and into the larynx. One of the authors instructed the subject while the other was responsible for the radiography. The 18 plates in each case had to be exposed very rapidly, as the bismuth did not adhere satisfactorily. All the exposures, therefore, were made within a few minutes. Ordinary plates were used without intensifying screens. A filter was placed against the subject's skin, and another filter of aluminum in front of the tube. The plates were carefully outlined in a viewing box, and each set of three carefully compared. The best plate from each series was outlined very carefully in ink, then diagrams were drawn from the plates directly, and at the same time sketches were made of the front view of the face for the various sounds.

Brunner and Fruehwald (1923, 1924) investigated the formation of vowels and consonants in deaf-mute children. They followed the procedure of Froeschels and Haudek and used their radiograms for normals. Tongue and lips were perfectly visible, but difficulty arose around the velum through peeling of the barium paste from the mucosal surface.

In 1924, Panconcelli-Calzia called the x-ray examination, as one of the appliances which expand the efficiency of our sense organs, one of the methods constituting the "iron reserve" of experimental phonetics.

Polland and Hála (1926) controlled the longer exposure time of 3 to 4 seconds by parallel exposures where the time was reduced to 0.1 or 0.05 seconds. It was shown that the articulation is not substantially modified during an emission of longer duration.

Gutzmann (1928) reiterated the statement of Scheier, that it is very easy to follow on the screen the movement of the soft palate when pronouncing the vowels, although when in a resting position that organ is almost invisible.

In 1929, a period began which one might call the golden age of application of roentgenography to phonetical investigation. The following years saw a number of projects in progress, based on extensive material, "to uncover secrets for us which had been hidden from the beginning of time," to follow the expression of Russell.

Russell (1928, 1929, 1931) included some 3,000 roentgenograms and accompanying measurements on over 400 subjects in his studies. In retrospect, he recalled that numerous such studies have been begun and been given up, because of danger, inconvenience, and expense. The idea of using edentulous subjects is of minor importance, bony surfaces having only a passing interest: the soft muscular surfaces are those which change the aspect of articulation. One should be able to make exact measurements of their dimensions. As to marker and nonmarker methods, each has its advantages. Without a marker one never knows when the tongue is convex and when concave. Without delineating with a marker the exact center of the tongue, no computations of the authentic cavity are possible. The median record shown by the roentgenogram is not only by far the most important but also the most enlightening. "The tongue creates most of the changes in the size of the vocal tract, and its movements are largely in the median plane. The extent to which it can alter the vocal cavity through the outer curvature and

lateral constriction of the tongue edges other than indicated by the x-ray photography is much more limited." On the other hand, a nonmarker technique recording only the misleading edges of the tongue is of no value. Partial length markers, or markers that are allowed to wobble back and forth along the tongue, give only an indication as to the general position of any part of the tongue. Some authors produced negatives so vague that they had to resort to rather crude pencil tracings made upon the originals.

Russell was inclined to proclaim that, in spite of important contributions (though all of them were made only on the basis of a limited number of experiments), roentgenography had until then yielded but little positive information which might be used in a definite manner, to such questions as: How do we talk? How do we produce subtle differences distinguishing different vowels? What makes the variety of quality heard in different voices? He spent over eight years in perfecting his x-ray technique. After giving a composite view of the x-ray laboratory as used in his experimental arrangement, he explained that the pictures have been so masked as to make the unessential part stand out whiter than the area involved in the vowel cavity itself. The real secret of a satisfactory picture is in the art of the development of the film, for which detailed instruction was given.

He found that barium is unsuitable, because the edges of the tongue would be accentuated as much as the center. Gold chains are not as opaque as lead. The link chain gags the subject. The smallest locket chain obtainable from a jeweler produced gagging. Extremely thin strips of gold have been abandoned, because their preparation needs great dexterity and new ones have to be provided in too rapid succession. His final marker was constructed in the following way: A very thin thread was prepared, like metallic rope about 0.5 mm. in diameter. The material was lead, gold-plated. It was flexible like a thread. It was not felt once it had been swallowed, adhering by capillary attraction to the center line of the tongue. In producing stereographic photographs, among others, he postulated an exposure of less than 1/100 of a second, unattainable at the time, to approximate the sound-pattern time for vowels.

Russell, in collaboration with Palomo (1934), asked for an exposure time reduced to at least 1/120 of a second for the study of normal speech. Since cineroentgenograms consisting of 1/120 of a second exposures were not feasible at the time, they found instantaneous exposure to be the next best technique. Synchronized sound picture records can be used to verify the final results.

Russell turned then (1934) to the study of consonants, which, being of very short duration, demand an automatic and very rapid technique. To take 17 exposures a minute (the most exposures possible at the time) turned out to be difficult and dangerous; besides, 17 exposures are not enough to reproduce the characteristic tongue position for the consonants. Instantaneous exposures were used, of 1/128 of a second, set off automatically by contact of the tongue with the roof of the mouth. The resulting photograph showed the tongue position at the point of maximum closure. A marker may facilitate the work of the "amateur radiographer"; otherwise, because of modern x-ray rapidity, it is to be discarded, as it gags and distorts.

Gutzmann (1930), after giving up intralingual injection of iodized oil ("iodipin") as too painful and as yielding only irregular shadows, covered the tongue with goldbeater's skin. This is a very tough membrane, and its fat component may be removed by washing in alcohol and ether. The skin was then spread on glass plates and dried with warm air. One piece of this skin was put for five months in iodized oil. It was 0.1 mm. thick. In this condition it was found unsatisfactory, even when coated again with iodized oil. Finally, the layer of iodized oil was reinforced with powder of barium sulfate, which, with the help of a few drops of a 5% solution of casein ("caseosan"), was painted on the skin, the latter being then spread on the surface of the tongue. Nose and lips were marked with a paste of barium sulfate and a 5% solution of casein. As the goldbeater's skin did not adhere well to the soft palate, a broad strip of peach skin was dehydrated in alcohol, coated with barium powder, and dipped for a second in water. This material adhered to the velum satisfactorily. Forty pictures were taken from the same subject, in sitting position, with the head pressed against the cassette. A person was chosen with as few teeth as possible; even then metal fillings were found disturbing. Later the iodized oil was omitted and only goldbeater's skin and barium sulfate were used.

Gutzmann was visited in 1929 by Russell, who reported that, by coincidence and independently, he too had used goldbeater's skin and iodized oil. Gutzmann found the photographs of Russell somewhat dull, as they were made with soft rays; many of Russell's pictures had to be retouched. Gutzmann worked at this time with an exposure of 0.2 seconds.

Gutzmann (1930) applied the cineroentgenographic method of Gottheiner and Jacobsohn in his investigations of speech sounds. The subject was a young man with a denture free of fillings, and with good speech technique—"a rare phenomenon." A full denture of aluminum was pointed out as a possible help, as this metal does not give a shadow. Barium with a 5% solution of casein and iodized oil served as contrast. He took 11 pictures per minute but postulated for exact phonetical work about 50, corresponding to an exposure time of about 1/100 of a second. He hoped to be able soon to study slow-motion pictures.

In his pursuit of the problem, Gutzmann (1931) applied an exposure of 30 seconds in about 750 pictures, using a film of 14-mm. length with an illumination of the single picture of about 1/150 seconds. He pointed out that these films will be of preeminent importance for the orator, the deaf-mute, the student of foreign languages, and the singer, and will be a great help in correction of speech defects and in experimental-phonetical work. Studying the films, he observed that the sum of all the movements can be comprehended in a satisfactory manner; but it is much more difficult to comprehend the special significance of the many simultaneous detail movements. The observer has to train himself to this kind of work. To help in this analysis it is useful to run the film many times without interruption, concentrating first on the movements of the lips until one learns them "by heart," and letting the other details follow. After this, several of the detail movements can be put together in a group to build up step by step the movements of the entire resonating tube. For teaching and experimental purposes, Gutzmann looked forward very much to the addition of the sound track.

Jones (1929) used a thin silver chain to mark the medial line of the tongue. A similar one was put through the floor of the left nasal meatus and passed over the soft palate into the pharynx. Each chain, including the small ball at its inner extremity, was about 10 cm. long and weighed only 0.5 gm., so that the distribution of weight per surface unit was very small. The exposure was 3/16 of a second. There was no distortion of utterance due to the chains. The contrast between the radiographs of some vowels and their "fanciful" representation in certain textbooks was striking.

Holbrook (1930), in cooperation with the roentgenologist van Nuys, put his 337 films in an orderly anatomical progression from each vowel to the next and planned to continue his systematic series "until I have enough to justify me in publishing articles or a book: X-Ray Studies of Speech Articulations:'"

Forchhammer (1930) found it useful to study the partial articulations which make up a vowel with the help of a systematic series of roentgenograms.

Huizinga (1930) worked with a bismuth paste as contrasting material. Much emphasis was placed on identical positions of the head.

Katzenstein (1930) placed the cassette parallel to the midline of the head and rested one edge on the clavicular fossa. In this way the larynx was included in the picture. Exposure of 1/50 to 1/60 of a second was advocated.

Parmenter, Trevino, and Bevans (1931) presented their method, emphasizing that, without markers, parts of the outline of the cavity do not appear. A fixed position of the head and the neck is necessary, so that the radiographs may be superimposed and compared. The film must be held in place parallel to the median section of the head and at right angles to the axis of the x-ray beam. A small gold chain placed exactly in the middle of the tongue was found necessary, since on the x-ray picture the outline of the chains and the middle of the tongue do not always fall together. The chain helps also to locate the floor of the mouth. Parmenter and his associates marked the hard palate with a strip of lead foil long enough to reach from the front teeth to the root of the uvula. Another strip of foil marked the median line of the back wall of the pharynx. A special headboard and a special cassette holder were constructed. Since only isolated and sustained sounds were examined, any exposure time which did not show movement of the organs was considered permissible; they adopted 6/20 or 7/20 of a second. The field surrounding the area to be investigated was shielded from the roentgen rays.

Parmenter and Trevino (1932), discussing the records of Russell, objected to the evidence revealing radical differences in the subject's posture. Holding the head in a fixed position is essential, as experimental tests have shown that changes in the position of the head entail changes in the position of the tongue.

The same author (1932), studying Spanish vowels, took the opportunity to emphasize that for each subject a sufficient number of pictures to discover his vowel positions is necessary. Each vowel was sustained for approximately three seconds, in the course of which time an exposure of 1/10 of a second was made, the three exposures being made on the same film.

Parmenter and Bevans (1933) reemphasized the necessity of studying the results of roentgenographic experiments comparatively, by superposition and by presenting them in a graphic form that can be easily comprehended. They described their technique of illumination and of making the laboratory tracings. The linguistic

background of the subject is important, and it is indispensable that the published results include a linguistic description.

Kelemen (1932) pointed out the difficulty encountered with the ventral (antero-posterior) exposure. Hardly any reliable information regarding the oral cavity can be gained in that manner. On the other hand, the pharynx, especially between the base of the tongue and the vestibule of the larynx, can be studied in many details with this exposure, while the larynx can be added, isolated from the shadow of the vertebral column, with the film lowered into the esophagus according to the technique of Réthi.

Menville and Ané (1932) painted a mixture of gum acacia and barium sulfate with a camel's hair brush, on the midportion of the tongue from base to tip and bisected the soft and hard palate with a coating of the same mixture. As saliva caused the mixture to spread after a while, glucose and bismuth subcarbonate was later used and proved to be an ideal marker. It cast a dense shadow, and the movements of the tongue and the saliva did not efface the preparation.

Weiss (1931), in a textbook chapter, summarized briefly the results of roentgenographic observation regarding the production of vowels.

Holbrook (1933) complained that subjects having regularity of anatomy and numerous other qualities required to furnish a sure basis for normal facts are rare. His 500 specimens of radiographs were displayed in a systematic series. The speaker had his right shoulder against the bottom of the cassette, with the median plane of the head and neck parallel to the film. Frequent spraying of the nose and the mouth with a compound of iodized sesame oil gave a dense but infinitesimally thin film over all exposed surfaces of the soft parts and thus disclosed the outline, or areas, or both. A chain cannot hold its intended position along the median line of the tongue. A leather helmet enables the speaker to hold or resume a given position, and an adjustable bracket holds the helmet at a given distance from the cassette. Attached to a vertical rod on the side of the cassette is a horizontal bracket, and on it an accurately adjustable slide with a rod pointing to the middle of the speaker's (closed) lips. A small rectangular cork block placed between the speaker's lips, to keep a constant distance between the incisors, was found generally to be a needless precaution. Exposure was about 0.05 second. Tracings were done on bluish cloth, tightly fastened over one film, on an electrically illuminated and electrically cooled calibrated viewing box. It was found that two-dimensional x-ray films reveal a "highly significant part of the truth."

Zwirner, in collaboration with Gottheiner (1933) who, together with Gutzmann, already (1929 and again 1931) had presented moving films of the speech organs, enumerated several difficulties in getting all the organs of speech onto a single film. When moving film is combined with reproduction of sound, the sound clarity is not disturbed markedly by the x-ray apparatus. He explained (1933) that simultaneous registration on the x-ray film and on the recording disc is a better demonstration of the movements of the organs of speech. He pleaded (1934) for fixation of the head, though this measure has a more or less disturbing influence on the speech. Concomitant movement of the head cannot be investigated in this way. Indentations of the tongue result in a crossing of the profiles. He reported experiments, not terminated, in collaboration with Brueck, to find a way to fill

the mouth of the subject with an adequate mass, hardening at the temperature of the oral cavity, to prepare a complete cast as help in x-ray photographs of the resonating tubes.

Zwirner (1935) postulated later the quantitative description and statistical evaluation of the results of the measurements concerning x-ray pictures of the articulation positions, if they are to be compared in a satisfactory manner.

Kelly and Highley (1934) wanted to photograph the posterior edge of the uvula. The sensitiveness of the average person does not allow the insertion of markers like chains, through the nostril, over the soft palate, and out through the mouth. Moreover, they found that the anatomy of the nasal structures makes it difficult to force a marker (metal chain or a specially treated string) to lie along the posterior edge of the uvula; such a marker persistently falls to one or the other side of the uvula. They prepared a paste of equal proportions of barium sulfate (to render the paste opaque) and acacia (to give adherence and moisture resistance), dissolved in lavoris* (to make it tolerable to taste). A median line 1/9 in. (0.28 cm.) to ¼ in. (0.64 cm.) wide was painted from the root of the tongue to its tip, and another along the palate from the tip of the uvula to the front teeth. To coat the posterior surface of the uvula, as far as the subject could stand the insertion, a spoon-like instrument on a long handle was applied. The head was fixed between the arms of an aluminum, horseshoe-shaped device with movable rods for adjustment. The exposure time was four seconds. Tracings were prepared from superimposed negatives.

Carmody (1936, 1937) studied the extensive material of Holbrook, after his death. Holbrook was accustomed to use no other check on the quality of vowels than his own ear, but in this he was most meticulous. His laboratory procedure was essentially the same as that of Parmenter and Trevino. Iodized oil U. S. P. (lipiodol*) was sprayed into the mouth, nose, and throat. He declared that while vowel articulation has been studied by roentgenography for many years, consonants have been neglected, partly because no tested procedure was available. Unlike the vowels, consonants often imply variable tongue positions. After analyzing the wide range of Holbrook's films, Carmody came to the conclusion that the secret lies not in tongue position but in its mode of contact with the palate, and in several aspects of shape and length of pharynx and larynx. Varying individual anatomy affects the articulation of any given sound, but the relative facts seem always to be the same. Later (1941) Carmody studied the pharyngeal articulation on 520 films of the Holbrook collection.

Hegedüs (1937) in his investigations of Hungarian vowels, pasted the anterior end of a thin gold chain to the chin; the free end was then swallowed with the help of water.

Fröschels, Hayek, and Weiss (1937), in their textbook chapter, discussed roentgen photography, skiagrams, polygrams, cinematography, and the addition of the sound track, in the study of phonetic movements. Concerning fixation of the head, they found that the naiveté of the articulatory movements is very much influenced by this procedure.

Chlumský, Pauphilet, and Polland (1938), investigating French vowels and semivowels, never used isolated sounds but sentences to assure a natural pro-

nunciation. An observer then caught the right moment for exposure upon utterance of the sound in question. Two kinds of radiographs were produced: one, in which the mandibular branches of the two sides covered each other; and another, in which they did not. A comparison between the two kinds is possible on the condition that the central ray is directed always through the same center, in this case the last superior molar.

Sovijaervi (1938) asked the subject to speak or sing the vowel for a certain time. When the phonation seemed loud enough and immobility was obtained, a sign was given and exposure followed, of a duration of about three seconds. Fixation of the head was omitted, because natural positions are influenced when the head is sustained. To avoid deviation from the natural conditions, no contrasting material was used. Even so, soft parts were seen with satisfactory clarity. Drawing and measuring were executed with the help of transillumination from the original x-ray negatives; exact outlines were obtained. In computing the roentgenograms the system of coordinates according to Zwirner was applied.

Curry (1938), in his study on the contralto voice, executed the following simultaneously: a radiograph; a synchronized cathode-ray oscillograph of the sound-wave form; a sound-on-disk recording; and an external motion picture of the facial expression.

Chiba and Kajiyama (1941), in their studies on the production and structure of the vowel, found that the most important data for the measurement of the vowel cavity are provided by x-ray photography. They adopted the gold chain method to mark the contour of the median section of the tongue and were able clearly to distinguish the elevation of this organ at the sides, from the depression in the middle. In this way they obtained their "radio-diagrams"—a diagram showing the median section of the vocal cavity, the raised sides of the tongue, and the teeth, traced from the x-ray photography.

Forchhammer (1942) published charts containing profile x-ray pictures of the vowel and consonant positions of the "world sound system."

Podvinec (1950) studied the movements of the soft palate with tomographic series of roentgenograms.

Delattre (1951) compared spectrograms with radiograms as one of the methods to determine the relation of formants to the position or movement of articulatory organs. In verifying some statements of his "Acoustic Vowel Charts," radiographs taken from Parmenter-Bevans were used.

PERSONAL TECHNIQUE

At the x-ray laboratory of the Department of Otolaryngology of the Massachusetts Eye and Ear Infirmary the following technique was found to yield satisfactory results.

The outline of the tongue, the roof of the mouth, and the pharyngeal wall were made visible by giving the subject a mixture of barium and water to which mucilage of acacia was added. With the use of the latter, the barium mixture can be made much denser with the same amount of water. Adhesion to the mucous membranes is very good. This mixture was applied by spatula, and the subject was asked to smooth over the surface with his tongue to get an even distribution.

It is essential that the head should be held in a natural speaking position and that the same position should be maintained throughout the entire procedure. At the same time, it is essential that the head should not be fixed, by a clamp or by any other device, in a strained position. In upright position, the head was placed against a headrest at the occipital region. The lower edge of the cassette rested against the side of the shoulder (against the deltoid) at a distance of about 22 cm. from the sagittal midline of the face. To prevent distortion caused by this considerable distance between the subject and the cassette, the x-ray tube was removed to 2 meters. In this way the distortion obtained was negligible. The central ray was directed through the crown of the first upper molar. The central ray being held constant, one was able to duplicate the work from day to day. As a further check to insure exact repetition of the position, a wooden throat stick was placed against the tip of the nose. A rotating tube was used, and the exposure time was 1/30 of a second, with 75 kilovolts and 480 milliamperes. The films used were Dupont with detail screens. Development was carried out under the usual standard vacuum technique. The tracings were made on transparent paper over a highly illuminated viewing box. The fix points, such as the occlusal surface of the upper incisors, the roof of the mouth, the position of the tip of the tongue, the dorsum and base of the tongue, and the position of the soft palate were sketched, together with the outlines of the posterior pharyngeal wall, the epiglottis, and the arytenoids. The accompanying tables offer a synopsis of the main technical points according to the different workers.

TABLE 1.—*Direction of Central Ray*

Central Ray Directed Against:	
Center of diaphragm of iris Polland and Hála	Between the upper and lower second molar Holbrook
Center of the cheek Fröschels and Haudek Gutzmann Hegedüs	Slightly above the level of the slot of the mouth and slightly behind the mandibular angle Eijkman
Level of the upper teeth Parmenter, Trevino, and Bevans	Mandibular angle Huizinga
Second upper molar Meyer	1 cm. below the mandibular angle Katzenstein Scheier
Last upper molar Chlumský, Pauphilet, and Polland	Body of the hyoid bone Grunmach

TABLE 2.—*Fixation of the Head*

Procedure omitted	Procedure applied
Barclay and Nelson	Barth and Grunmach
Curry	Chlumský, Pauphilet, and Polland
Gutzmann	Eijkman
Katzenstein	Haudek-Fröschels
Meyer	Holbrook
Scheier	Kelly and Highley
Schilling	Parmenter, Trevino and Bevans
Soevijaervi	Polland and Hála
	Russell
	Zwirner

TABLE 3.—*Use of Contrast Materials*

Use rejected	Lead foil
Curry	Grunmach
Fröschels, Haudek, and Weiss	Parmenter, Trevino, and Bevans
Navarro	Tin foil with barium
Scheler	Chiba and Kajiyama
Soevijaervi	Metal chain
(Edentulous subject: Scheler)	Barth and Grunmach
(Denture with metal fillings: Gutzmann)	Chiba
Bismuth or barium (paste or powder)	Chiba and Kajiyama
Barclay and Nelson	Hegedüs (sometimes omitted)
Chlumský, Pauphilet, and Polland	Jones
Eijkman	Meyer
Fröschels and Haudek	Parmenter, Trevino and Bevans
Gutzmann	Russell (later omitted)
Huizinga	Goldbeater's skin with iodized oil
Kelly and Highley	Gutzmann
Menville and Ané	Russell
Polland and Hála	Peach skin with barium powder
Iodized oil	Gutzmann
Holbrook (spray)	Hardening mass in oral cavity
Huizinga (swallowing)	Zwirner
Iron powder (paste with syndetikon)	Copper wire cross on frame
Meyer	Eijkman

COMMENT

"Neither the naked hand nor the understanding, left to itself, can effect much. It is by instruments and helps that the work is done." (Bacon)

Experimental elimination of an organ in order to study its role in the organism is hardly feasible in the case of the anatomical elements used in articulation, but accidental and operative mutilations have given plenty of opportunity for this line of observation. Speech without the most important organ of articulation, the tongue, was discussed by Twistleton, as early as 1873, in an extensive monograph. Defects of the cheeks or of the lateral pharyngeal wall offered the possibility for direct observation. To represent the position of the phonatory organs through such windows was the classic method of illustration (Helmont, 1667; Wilkins, 1668; Kempelen, 1791). With the advent of roentgen rays, roentgenography became the sovereign method to analyze conditions originating in loss of one of the organs. Knowledge as to speech without larynx was substantially helped in diagnosis and treatment by x-ray films, as was also the study of faulty arrangement of speech organs, or improper tongue placement caused by hiatuses in the dental alignment (sigmatism). Analysis of a number of other speech defects was furthered by roentgenography.

The shape and size of the vocal passages, when set to produce the different sounds, were roughly approximated by the tubes of Kratzenstein (1782). The real advance from philosophical speculation to experimentation came with Kempelen (1791) (Dudley and Tarnoczy).[1] Mechanical elements were constructed and manipulated to simulate the essential parts of the human vocal system; the speech machine had to be constructed on factual knowledge.

1. Dudley, H., and Tarnoczy, T. H.: The Speaking Machine of Wolfgang von Kempelen. J. Acoust. Soc. Am. **22**:151-166 (March) 1950.

Experimental work on phonatory movements did not begin until the middle of the 19th century. It continued towards its goal with acceleration, culminating in the methods to make speech visible in one or another way. Prejudices had to be overcome. Jones complained about the superior attitude toward experimental work of some phoneticians, "to whom phonetics is a matter of faith rather than of experiments." After seeing the talking film of Menzerath, combined with the sound track in 1935, the reviewer of the congress was convinced that speech consisted of a continuous complexity of almost countless movements; there were no "speech positions," no "on glides" and no "off glides" or any of the other familiar clichés of nonexperimental phonetics: he found that by this film "the entire physiology of the present day was swept away at the first glance."

Certain key concepts appear at first vague and indefinite, and remain so until development of the necessary equipment makes it possible for them to be checked against observational data (Békésy-Rosenblith).[2] The main concepts gained by early investigators, partly by self-observation, could be confirmed by roentgenography, but many details had to be corrected after exact testing. An example might be cited: Merkel, in the mid-19th century, conceded a considerable variation latitude of articulatory positions to the single vowels. This latitude was step by step narrowed down until surprisingly exact and constant boundaries were found, inside of which these movements are carried out.

There are important gaps in x-ray observation to be filled in at a future time. Many details escape with the exclusive study of profile structures, as when the torus of Passavant is observed but not the protrusion of the lateral pharyngeal walls, which aid, from the sides, in the closure of the nasopharyngeal cavity (Joachim).[3] There is room for technical improvement along this line, for example, in the anteroposterior larynx picture obtained by intraesophageal films according to Réthi,[4] or by wider application of planigraphy.

After Scheier emphasized their usefulness, comprehensive roentgenograms, showing in a single picture the total, integrated vocal mechanism from the diaphragm to the lips, were the object of several studies. Otherwise the main object of observation, apart from the resonating tube, was the larynx. At this point it might be useful to recall that speech and voice quality differences, generally ascribed to the modificator influence of the cavity above the vocal cords, were traced by Russell, with the help of roentgen rays, to the interior of the larynx.

Crandall complained of the curious fact that most of the data on the shape of the oral cavity, on the positions of the tongue, etc., in producing the different vowel sounds, have been obtained by students of phonetics. For finer differences, one has to rely on other sources than those given in the literature of anatomy, in which determinations of the actual volumes of mouth and pharynx, for example, are entirely lacking. One approach through phonetical x-ray studies might well be to fill the gaps in general anatomical and physiological knowledge.

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2. Békésy, G., and Rosenblith, W. A.: The Early History of Hearing, J. Acoust. Soc. Am. **20**:727-748 (Nov.) 1948.

3. Joachim, O.: Contribution to the Physiology of the Soft Palate, Arch. Otol. **18**:226-234, 1889.

4. Réthi, A.: Meine neue Methode bei der Röntgendarstellung des Kehlkopfes und der Luftröhre, Ztschr. Laryng. **6**:27-33, 1914.

The entire course of development of the x-ray technique in its application to articulatory movements shows periods of "spurts," with several important projects in progress simultaneously, alternating with periods of lull. *Habent sua fata libelli:* this was substantiated several times during the course of these investigations. Panconcelli-Calzia's textbook on roentgenology of human phonetical movements, in galley proofs July, 1914, was printed in only three provisional copies: these survived the first World War only to be destroyed in the course of the second. The results of the extensive investigations of Barclay were lost by theft. Barth-Grunmach and Meyer published, independently of each other and in the same year, almost identical methods. Gutzmann and Russell, independently again, conceived at the same time the same idea—to use goldbeater's skin as contrasting material.

The dividing line between the "acousticist" and "phoneticist" used to be sharp, so much so that Stumpf, in 1926, spoke of a mutual mistrust, even a want of confidence in each other's methods of investigation. Roentgenography did much to break down this barrier. Simultaneous production and analysis of the visual and the auditive picture, by combining cinematography with the sound track, paved the way which led to the establishment of the mushrooming "audiology" departments, and welded the dual approach into a fertile and promising unit.

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LARYNGEAL MANIFESTATIONS OF TABES DORSALIS

Review of the Literature and Report of Eleven Additional Cases

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RARE clinical syndromes go through cyclic periods of familiarity to physicians. When once they have been described by an alert observer, examples are reported by others, a group of papers is published and the syndrome becomes for a time so well known and almost commonplace that further papers cease for a time to appear. Then a new generation of physicians grows up, so occupied with current medical journals as to have little time for the older literature, and the syndrome is temporarily forgotten, only to be rediscovered and redescribed.

Such a syndrome is the laryngeal manifestations of tabes dorsalis. Known for nearly a century to exist, they are still so unfamiliar to most neurologists and laryngologists as to justify a review of the literature and the report of an additional series of cases.

REVIEW OF THE LITERATURE

Clinical Aspects.—The first description of tabetic involvement of the larynx is attributed to Féréol,[1] who in 1868 reported three examples of "laryngeal crises" but provided no information as to the presence or absence of laryngeal paralysis. In 1879, Charcot[2] observed two tabetics who suffered from attacks beginning with burning or tickling sensations in the lower larynx, followed by dry cough, loss of consciousness, and convulsions. The attacks were short, and the patients were apparently well afterward.

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1. Féréol, L. H. F.: De quelques symptomes visceraux et en particulier des symptomes laryngobronchiques de l'ataxie locomotrice, Soc. med. hóp. Paris **5** (Ser. *2*):82, 1868; he also refers to a similar case (apparently not published) observed by Cruveilher in 1825.

2. Charcot, J. M.: Ataxie locomotrice (série tabétique): crise laryngée, arthropathies; sclérose latéral amyotrophique, Gaz. hóp. Paris **52**:3, 1879.

In short order there appeared reports of similar cases by Krishaber [3] (1880), Cherchevsky [4] (1881), and Lhoste [5] (1882).

In 1885, the elder Fournier, utilizing these communications and his own vast experience, devoted to the laryngeal manifestations of tabes dorsalis a 22-page chapter of his "Leçons sur la période préataxique du tabès d'origine syphilitique," which for clarity of clinical observation and expression can hardly be improved upon today.[6]

Fournier classified the laryngeal manifestations of tabes dorsalis into five groups: (1) laryngeal paralysis; (2) spasmodic cough; (3) aphonic spasm; (4) suffocating attacks; (5) fatal "apoplectiform" attacks of apneic spasm. The last four of these subdivisions represent various gradations of severity of laryngeal crises, which Fournier recognized might occur with or without actual laryngeal paralysis.

He reported five examples of recurrent laryngeal nerve paralysis in tabetics and felt that this phenomenon (and that of laryngeal crises as well), like extraocular muscle palsies, was characterized by its early, often "preataxic," occurrence and usually by spontaneous disappearance after a period of several months, without apparent relationship to specific treatment (with mercury).

Spasmodic cough, according to Fournier, occurs in attacks of varying degrees of severity, sometimes so intense as to resemble the cough of pertussis and, as in pertussis, sometimes accompanied by or ending in vomiting. There may or may not be a premonitory sense of tickling, tingling, burning, or constricting sensations in the larynx. The cough is dry and unproductive. It is unrelated to any evidence of upper respiratory infection and is remarkable for its long duration (months or years) and its refractoriness to medication of any sort. It is "nervous," obviously spasmodic, and often apparently precipitated by a trigger mechanism, such as exposure to a draft of air, contact of the skin with a cold object, very hot or cold foods, or even emotional upsets.

Fournier described aphonic spasm as follows: At intervals, abruptly and without warning, the patient is unable to speak at all, or at least he cannot speak in his ordinary tone of voice. The voice may be "muffled, hollow, false, discordant as if the two vocal cords no longer vibrated in unison." With or without this phenomenon, there may suddenly occur a "sort of inspiratory crow." Sometimes associated are bizarre modifications of respiratory rhythm with inspiratory spasmodic convulsive jerks, as if there were "convulsions of the diaphragm." The prognosis is grave, and in a severe attack of repiratory distress such aphonic spasm may even lead to death.

In severer form suffocating attacks may occur, with severe dyspnea or even apnea. Again the onset is sudden and unpredictable, even occurring during sleep. There may be a brief aura of laryngeal paresthesias—burning, tickling, or constriction. The episode may attain maximum intensity in an instant, with extreme

3. Krishaber, M.: Du spasme laryngé dans l'ataxie-locomotrice, Ann. mal. oreille et larynx **6**:249, 1880.

4. Cherchevsky: Contribution à l'étude des crises laryngées tabétiques, Rev. méd. **1**:541, 1881.

5. Lhoste, L. C.: Étude sur les accidents laryngés de l'ataxie locomotrice progressive, Thesis, (#226), Paris, 1882.

6. Fournier, A.: Leçons sur la période préataxique du tabes d'origine syphilitique, Paris, Masson, 1885, pp. 239-260.

anguish, dyspnea, whistling respirations, a sense of strangulation, engorgement of the face, and cyanosis. Such patients may fall unconscious, and epileptiform convulsive movements simulating grand mal may occur. Then, after a few moments, and as if by magic, the crisis terminates.

In extreme form, if such crises as have just been described last for more than a few moments, the heart beat weakens and the patient dies suddenly. Fournier had seen two such cases, which he called "fatal 'apoplectiform' attacks of apneic spasm." For such severe laryngeal crises, said Fournier, emergency tracheotomy is lifesaving.

In speculating as to the mechanism of laryngeal crises, Fournier was certain that they were not related to paresis or paralysis of the vocal cords. Objective evidence for this opinion, he said, does occur in some such cases, but in the majority it is absent. Moreover, he observed that in laryngeal paralysis due to other causes such crises do not occur. Fournier accepted the hypothesis of laryngeal spasm but had no idea as of the mechanism that produces it.

In 1895, both Herms [7] and Zschirnt,[8] and in 1898 Cadel,[9] attested interest in the subject with short monographs devoted to laryngeal disturbances in tabes dorsalis, each of which reviewed the literature but added little to the clinical description of Fournier.

Numerous reports of one or more cases of laryngeal crisis, laryngeal paralysis, or the two together, and of all grades of severity, have appeared in the literature. Both Hawkins [10] (1895, one case) and Lockard [11] (1903, three cases) emphasized that paralysis, when present, usually begins with or may be limited to the posterior cricoarytenoid muscle and is usually, if not invariably, unilateral. Subjective symptoms may be completely lacking and the paralysis discovered only on routine laryngoscopy.

Among other case reports, with or without discussion of the literature, are those of Adams,[12] Lewandowsky,[13] Gregory,[14] Kriskemper,[15] McFadden,[16] Fox,[17] Schüler,[18] Brookfield,[19] Strauss,[20] Terracol and his associates,[21] Bock,[22] Chavany and

7. Herms, F.: Beitrag zu den Störungen des Kehlkopf bei Tabes, Berlin, B. Paul, 1895.

8. Zschirnt, A.: Larynxkrisen bei Tabes dorsalis, Erlangen, A. Vollrath, 1895.

9. Cadel, A.: Les paralysies laryngées du tabes, Lyon, 1898.

10. Hawkins, F.: Case of Bilateral Paralysis of the Crico-Arytenoidei Postici Associated with Tabes Dorsalis; Asphyxia; Tracheotomy, Recovery, Lancet **1**:1373, 1895.

11. Lockard, L. B.: Laryngeal Paralysis as a Primary Symptom of Tabes Dorsalis, Ann. Otol. Rhin. & Laryng. **12**:50, 1903.

12. Adams, F. M.: Laryngeal Paralysis in Tabes, Providence M. J. **17**:153, 1916.

13. Lewandowsky, M.; Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Springer-Verlag, 1911, Vol. 2, p. 983.

14. Gregory, L. T.: Laryngeal Crisis with an Unusual Complication: Report of a Case, J. A. M. A. **74**:793 (March 20) 1920.

15. Kriskemper, C.: Plötzlichen Atemstillstand bei Tabes dorsalis, Med. Klin. **23**:436, 1927.

16. McFadden, J. F.: Bradyapnea or Transitory Bilateral Vagus Paralysis in Tabes Dorsalis, M. Clin. North America **11**:531, 1927.

17. Fox, N.: Laryngeal Crises of Tabes, Illinois M. J. **54**:66, 1928.

18. Schüler, A.: Über einen Fall von vorübergehender Atemlähmung bei Tabes Dorsalis, Klin. Wchnschr. **8**:74, 1929.

19. Brookfield, R. W.: Paralysis of Last Four Cranial Nerves in Tabes Dorsalis, Lancet **1**:1287, 1930.

(Footnotes continued on next page)

Daum,[23] and Bumke and Foerster.[24] Both McFadden and Strauss emphasized the fact that apneic crises in tabes dorsalis are often preceded by the administration of morphine. Bock pointed out that even in severe crises cardiac rate and rhythm are usually undisturbed, an important point as it relates to vagotonia and the autonomic nervous system. In 1930, Schiff [25] was able to collect 110 cases of laryngeal crises, in exactly half of which (55 cases) there were also laryngeal paralyses. He emphasized the lifesaving value of tracheotomy in severe laryngospasm, especially when vocal cord paralysis is present.

We have made no attempt to gather from the literature all the reported cases,[26] which probably now number over 300 and include some 20-odd examples of death in apneic crises.

Several workers have studied the incidence of laryngeal involvement in tabes dorsalis by means of routine laryngologic (in some instances direct laryngoscopic) examination. Semon [27] found some form of laryngeal paralysis in 14 of 100 patients with tabes dorsalis. Lockard [11] also cited Gerhardt (original reference not located) as having observed laryngeal paralyses in 17 of 122 tabetics. Combining these two series, Lockard remarked that in the 31 instances reported the paralyses were as follows: unilateral posticus, 14; bilateral posticus, 8; unilateral recurrent laryngeal, 5; bilateral recurrent laryngeal, 1; complicated, unclassified, 3.

It is probable that the 17 cases of Gerhardt just cited are also included in a larger number reported by Dorendorf,[28] who in 1903 reviewed 245 cases of tabes dorsalis from Gerhardt's clinic in Berlin. He found that in 12% of the cases there was vocal cord paralysis and in 3% of the total laryngeal crises. Jerky (ataxic) movements of the cords on phonation were observed in 4% of all the cases. The latter was interpreted as a sign of impending vocal cord dysfunction.

In 1906, Greene [29] reviewed 60 cases of tabes dorsalis, in 9 (15%) of which there were laryngeal complications (nature not stated), in 6 (10%) undoubted

20. Strauss, H.: Zur Kenntnis der "Oblongatakrisen" mit Atemstillstand bei Tabes dorsalis, Deutsche med. Wchnschr. **59**:406, 1933.

21. Terracol; Sagol, H.; Bert, and Cadilhac: Diplégie laryngée (paralysie des dilatateurs chez un tabétique), Arch. Soc. sc. méd. et biol Montpellier **14**:355, 1933. Terracol, J.: Les paralysies laryngées périphériques: État neuro-laryngologique de la question, Rev. laryng. **52**:231, 271, 1931.

22. Bock, H. E.: Über Atemkrisen bei Taboparalyse: Beitrag zum Mechanismus angiospastischer Insulte und des paralytischen Anfalls, Zentralbl. inn. Med. **55**:961, 977, 1934.

23. Chavany, J. A., and Daum, S.: Tabes et perturbations neuro-végétatives paroxystiques, à propos des crises laryngées et de certaines crises vaso-motrices de l'extrémité céphalique, Presse méd. **43**:1505, 1935.

24. Bunke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Springer-Verlag, 1935, Vol. 12, p. 553.

25. Schiff, L.: Chirurgische Zufälle bei tabischer Kehlkopfkrisen, Deutsche Ztschr. Chir. **226**:66, 1930.

26. The Index Catalogue of the Library of the Surgeon General's Office, U. S. Army, Series 3, 1920, Vol. 2, p. 138, lists at least 18 titles of case reports of laryngeal involvement in tabes dorsalis not included in the present references.

27. Semon, F.: Die Nervenkrankheiten des Kehlkopfes und Luftröhre, in Heymann: Handbuch der Laryngologie und Rhinologie, Vienna, A. Hölder, 1898, Vol. I, p. 587.

28. Dorendorf, H.: Kehlkopfstörungen bei Tabes, nach dem Material der Gerhardtschen Klinik, Berlin, O. Enslin, 1903.

29. Greene, D. C., Jr.: A Study of the Larynx in Tabes, Boston M. & S. J. **154**:97, 1906.

abductor paralysis of one or both vocal cords, and in 7 (12%) laryngeal crises (in 3 of which there was no evident paralysis of either cord).

In 1907, Gräffner [30] reported that in a series of 206 tabetics the amazing number of 88 (42.7%) had laryngeal symptoms of some sort, and no less than 55 (26.7%) had vocal cord paralysis. The paralysis was of the following types: left posticus, 28; left posticus plus left "internus," 8; right posticus, 7; both postici, 8; left recurrent, 3; bilateral recurrent, 1. Of the 55 with laryngeal paralysis, 26 also had laryngeal crises, and 20 of the 26 had gastric crises in addition. He mentioned that tremors of the vocal cords are an early sign of vocal cord dysfunction, and although such tremors may also occur in other diseases, such as hysteria, paralysis agitans, senile dementia, alcohol and lead intoxication, multiple sclerosis, and pseudobulbar palsy, they are much commoner in tabes dorsalis.

New and Childrey [31] in 1932 agreed with the observations of Burger, Gräffner, Greene, and others that laryngeal crises might occur in tabetics with apparently normal vocal cords.

We have been unable to find more modern reports of routine laryngoscopic study of tabetics, and in view of the apparent rarity of laryngeal involvement, as exemplified by our own material (presented below) and by more recent literature, we feel that the entire subject is worthy of reexamination.

Approaching the subject from the reverse point of view, i. e., the etiologic background of a series of cases of laryngeal paralysis, New and Childrey,[31] and Smith, Lambert and Wallace,[32] agree that tabes dorsalis is a rare cause. The former workers identified only 9 of 217 cases of "vocal cord paralysis," and the latter only 2 of 235 cases of "recurrent laryngeal nerve paralysis," as due to tabes dorsalis. Of these 11 cases, in 10 the paralysis occurred in males, and in at least 6 it was bilateral. It is not clear that these workers have taken into account the possible frequency, pointed out above, of symptomless posticus paralysis.

Pathogenesis of Laryngeal Manifestations in Tabes Dorsalis.—The literature contains meager information regarding the pathologic processes involved in the production of laryngeal paralysis of syphilitic origin, aside from those instances in which the paralysis is directly due to the pressure of a mediastinal mass (usually aneurysm) or the presence of a local gummatous infiltration in the region of, or directly involving, the vocal cords. (Gummatous laryngitis is outside the scope of this discussion.)

Reporting the necropsy findings in a patient who during life suffered from a combination of laryngeal crises and abductor paralysis, Davis [33] described cell atrophy without evidence of inflammation in the nucleus ambiguus and in the motor nuclei of the 9th, 10th, and 11th cranial nerves. There were also atrophy and demyelination of the affected vagus nerve. In discussing Davis' paper, Watson-Williams suggested the possible extension (not actually demonstrated) of the

30. Gräffner, H.: Studien über Tabes dorsalis mit besonderer Berücksichtigung der Kehlkopfsymptome (221 Fälle), München med. Wchnschr. **36**:1775, 1907.

31. New, G. B., and Childrey, J. H.: Paralysis of the Vocal Cords: A Study of 217 Medical Cases, Arch. Otolaryng. **16**:143 (Aug.) 1932.

32. Smith, A. B.; Lambert, V. F., and Wallace, H.: Paralysis of the Recurrent Laryngeal Nerve: A Survey of 235 Cases, Edinburgh. M. J. **40**:344, 1933.

33. Davis, E. D. C.: Specimens from a Case of Laryngeal Crisis with Abductor Paralysis, Proc. Roy. Soc. Med. (Laryng. Sect.) **9**:95, 1915-1916.

degenerative process to the cardioinhibitory nuclei, in order to account for the resting tachycardia frequently observed in such patients.

Several interesting observations and speculations were made by Burger [34] in a lengthy monograph on laryngeal disturbances in tabes dorsalis published in 1891. He stated that the medulla is often involved in tabes dorsalis and that among the bulbar nerves the vagus and accessory are the most frequently affected. The involvement of the laryngeal nerves is a motor paralysis with coordination abnormalities (laryngeal ataxia). The motor paralysis may be completely asymptomatic. The posterior cricoarytenoid muscle is most frequently involved. Burger doubted that there is an isolated adductor paralysis, although temporary functional adductor paralysis may occur. He believed that the superior laryngeal nerve is rarely involved and that when it is the involvement is always in conjunction with that of the inferior (recurrent) laryngeal nerve. The median position of the cords in cases of bilateral vocal cord paralysis is the result of secondary contracture of the adductors attendant upon primary paralysis of the posterior cricoarytenoid muscle. Laryngeal crises may occur with or without paralysis of the cords. Crises result from excitation of the sensory fibers of the laryngeal nerves, usually associated with increased reflex stimulation of the centers of the adductors of the cords. The stimulus which produces the attacks arises from chronic degeneration of the nerve tissue located in the medulla. The anatomic picture of tabetic laryngeal paralysis is a degeneration of the nucleus of the vagus-accessory nerves or fibers in the medulla, together with a degenerative atrophy of the vagus and recurrent nerves.

Rebattu and Mounier-Kuhn [35] stated: "In laryngeal paralysis of bulbar origin, syphilis plays a primary role because it is responsible for most of the focal lesions by hemorrhage and especially by softening due to syphilitic arteritis." They described the postmortem findings in a case of vocal cord paralysis in a tabetic who died after a tracheotomy had been performed. Here they found "histologically an active inflammatory process without sclerosis or definitive ischemia lying in the retro-olivary region in contact with the radicular intrabulbar fibers of the pneumogastric between their nuclear origin and bulbar emergence."

Fournier [6] did not describe the fundamental pathologic process in vocal cord paralysis due to syphilis, but he believed that it is "sometimes a question of a simple neuritis." He believed that the abductors were the first muscles affected. If the neuritis was bilateral, the two cords remained in contact with each other. The voice was not affected thereby, but there was severe inspiratory stridor, especially noticeable upon mild exertion. Later, as other intrinsic muscles of the larynx became involved, the cords assumed a midposition (between complete abduction and adduction) called the "cadaveric position." The final event was, he believed, an atrophy of all the muscles controlling the cords and even of the cords themselves.

Grabower (cited by Greene [29]) found at autopsy a degeneration in the motor fibers of the vagus which supply the recurrent laryngeal nerve. This degeneration affected not only those fibers supplying the posterior cricoarytenoid muscle but also those supplying the other intrinsic muscles of the larynx. By actual count he found that nerve fibers in the posterior cricoarytenoid muscle were fewer than those in the

34. Burger, H.: Die laryngealen Störungen der Tabes dorsalis, Leiden, E. J. Brill, 1891.

35. Rebattu, J., and Mounier-Kuhn, P.: Syphilis du larynx et paralysies laryngées, Oto-rhino-laryng. internat. **20**:723, 1936.

other groups. He believed that, since the nerve fibers to all the muscles supplied by the recurrent laryngeal nerve were affected but only the abductors (posterior artenoid muscles) were paralyzed, this must indicate that the abductors were a "weaker group" with respect to nerve supply and that they degenerate first. This is a hypothetical explanation of "Semon's Law" [36] (that the earliest sign of injury to the recurrent laryngeal nerve is loss of ability to abduct the vocal cords and that loss of adduction appears only when the nerve is totally inactivated).

New and Childrey [31] stated that the abductors are analogous to extensors and are newer developmentally. Their fiber bundles are not so large as those of the adductors and are hence more vulnerable to destructive processes.

Semon and Watson-Williams [37] reiterated the greater vulnerability of the abductors and added that the spastic phenomena (jerky movements of the cords) may be due to increased irritability of the adductor centers. The peripheral stimulus conducted along the centripetal fibers of the superior laryngeal nerve to those centers (which, according to the above hypothesis, are subject to increased irritability) does not start a mere cough (as normally) but produces cough, glottis spasm, and generalized laryngeal convulsion (i. e., crises). They also state that in tabes dorsalis in association with abductor paralysis the pulse is often persistently elevated. This is thought to be due to the fact that the inhibitory nerve of the heart, like the motor nerve of the larynx, is derived from the accessory nucleus.

From The Johns Hopkins Hospital, Langworthy [38] reports the necropsy findings in a fatal case (not included in the present series). The patient was a 58-year-old Negro man with syphilis in whom, about two months before death, hoarseness and dysphagia developed. There were neurologic signs pointing to bilateral involvement of the glossopharyngeal and vagus nerves (including complete bilateral paralysis of the vocal cords) and weakness of the right spinal accessory nerve. At necropsy, scattered perivascular round-cell infiltration throughout the medulla, chromatolysis of the autonomic and somatic motor cells of both glossopharyngeal and vagus nuclei, "loss of medullated fibers in the region of the dorsal nucleus of the vagus and medial to this area," and lymphocytic infiltration around the glossopharyngeal and vagus rootlets after their emergence from the medulla were observed. There is no record of microscopic study of the nerves themselves, but the evidence seems clear that in this case, at least, the pathologic lesion was central rather than peripheral.

Neuroanatomy and Physiology of the Larynx.—The complicated musculature of the larynx is supplied by the 10th (vagus) nerve, which is the longest and most complicated of the cranial nerves. Much remains to be learned of its physiologic function, especially with respect to its visceral components. The larynx serves, however, as an accurate mirror of gross interruption of its function.

The origin and function of the pharyngeal and laryngeal branches of the vagus nerve, the effects of complete paralysis of the vagus, and the laryngeal symptoms of various forms of laryngeal paralyses are shown in Tables 1, 2, and 3, modified from

36. Semon, F.: Proclivity to Disease of Abductor Fibers, Arch. Laryng. **2**:197, 1881.

37. Semon, F., and Watson-Williams, P., in Allbutt and Rolleston: System of Medicine, London, The Macmillan Company, 1910, Vol. 4 (Pt. 2), p. 280.

38. Langworthy, O. R.: A Case of Bilateral Paralysis of the Glossopharyngeal and Vagus Nerves in Man, Bull. Johns Hopkins Hosp. **50**:76, 1932.

TABLE 1.—*Origin and Function of Pharyngeal and Laryngeal Branches of Vagus*
(After Wilson and Bruce [39])

Branch		Origin and Course	Function	Supplies (Area or Muscle)	Functions
Pharyngeal		From nodose ganglion at jugular foramen; unites with twigs of 9th nerve and superior cervical sympathetic ganglion to form pharyngeal plexus	Motor	Constrictors of pharynx	Narrows pharynx
				Palatoglossus	Lifts tongue; narrows fauces
				Palatopharyngeus	Narrows fauces; shuts off nares
				Levator palati; azygos uvulae	Moves soft palate; helps to shut off nares
Superior laryngeal	Internal laryngeal	As for pharyngeal	Sensory	Mucous membrane of larynx, glottis, and base of tongue	
	External laryngeal		Motor	Cricothyroid	Tightens cords
Recurrent (inferior) laryngeal	Right	From vagus in neck, hooks around subclavian artery, turns upward between esophagus and trachea, enters larynx from below; en route sends branches to deep cardiac plexus, inferior cervical ganglion, trachea, esophagus, and inferior constrictor of pharynx	Motor	Thyroarytenoids	Relaxes cords
				Cricoarytenoid (posticus)	Abducts cords
				Cricoarytenoid (interalis)	Abducts cords
				Arytenoid (oblique)	Serves as sphincter of upper larynx
				Arytenoid (transverse)	Serves as sphincter of glottis
	Left	From vagus in thorax, hooks around ascending aortic arch; thereafter as for right			

TABLE 2.—*Effects of Complete Paralysis of Vagus*
(After Wilson and Bruce [39])

		Structure Affected	Effects
Complete paralysis of vagus	Unilateral	Palate	Soft palate droops on affected side, does not rise with phonation
		Pharynx	"Curtain movement" of posterior pharyngeal wall on saying "a-a-a-ah" or gagging—i. e., unopposed contraction of sound constrictor pulls its weak partner across midline; anesthesia of affected side
		Larynx	Vocal cord assumes intermediate or cadaveric position; voice husky, singing impossible; coughing ineffective; articulation of consonants not disturbed; dyspnea and stridor absent; laryngeal anesthesia on paralyzed side
		Other local	Produces anesthesia of external auditory meatus and back of ear
		Visceral	Causes bradycardia or tachycardia, otherwise indefinite
	Bilateral	As above	Produces above results, plus extremely "arduous and slow" respiration; rapid and irregular cardiac rate; dilated and atonic stomach and esophagus; death

TABLE 3.—*Laryngeal Symptoms of Various Types of Paralyses*
(After Wilson and Bruce [39])

Branch Affected			Symptoms
Superior laryngeal	Rare except from section (as accidentally at operation or attempted suicide)		Laryngeal anesthesia; cricothyroid palsy with affected cord no longer tense, but slack on phonation, flaps up and down with respiration; voice weak and soon tired; if bilateral, voice hoarse and deep
Recurrent laryngeal	Complete paralysis	Unilateral	Paralysis of both abduction and adduction, affected cord in cadaveric position, i. e., midway between abduction and adduction; does not move with phonation or respiration; on phonation, unaffected cord crosses over to join; voice hoarse but not lost
		Bilateral	Both cords immobile in cadaveric position; aphonia; dyspnea; inspiratory stridor
	Partial paralysis	Due to organic causes	Abduction (cricoarytenoid posticus) affected first; if unilateral, usually symptomless; if bilateral, cords come together during phonation, voice therefore unaffected; in inspiration, cords do not move outward but may be "aspirated" together, producing severe dyspnea and stridor; if lesion progressive, thyroarytenoid (which relaxes cord when acting alone, but sharpens and tightens edge when acting with cricothyroid) and lateral cricoarytenoid (adductor) affected in order (Semon's law)
		Hysterical	Adductor paralysis (bilateral)

Wilson and Bruce.[39] It should be at once noted that both pharyngeal and laryngeal branches originate from the vagus nerve in close apposition, i. e., from the nodose ganglion at the jugular foramen, and that a lesion at or above this point (e. g., in the nucleus ambiguus) might be expected to involve both. Since this is not the case in the patients with tabes dorsalis reported here or in the literature, in whom pharyngeal paralysis is conspicuous by its absence, one is perhaps justified in believing that in tabes dorsalis the lesion is peripheral rather than central.

The internal branch of the superior laryngeal nerve is the sensory nerve of the larynx; the external branch supplies a single pair of muscles, the cricothyroid. Both in our own tabetic patients and in those described in the literature there has been inadequate examination of sensory laryngeal function, and we have not observed in our own cases, and have not found in those of other investigators, examples of isolated unilateral or bilateral cricothyroid paralysis. If this is actually the case, it would then seem to follow that in most tabetics with laryngeal paralyses the lesion is peripheral to the nodose ganglion, since this area is thought to be the origin of the superior laryngeal nerve.

Further support is lent to the theory of peripheral location by the fact that in the large majority of tabetics with laryngeal paralyses the course of events follows Semon's law: The first evidence of trouble is cricoarytenoid (posticus) paralysis, which if unilateral is usually symptomless. Obvious damage may, and apparently usually does, stop at this point. If progressive, adductor (cricoarytenoid lateralis) and sphincter (arytenoid) paralyses appear as later phenomena.

On the other hand, in four of our own cases and in some of those reported in the literature, complete bilateral posterior cricoarytenoid paralysis has occurred more or less abruptly with "apoplectiform" suddenness, necessitating emergency tracheotomy to prevent death from strangulation.

It is clear, moreover, both in our own cases and in those of others, that the lesion in tabes dorsalis is intrinsic in the medulla or the nerve itself and is not due to extrinsic pressure. In none of our cases was there any clinical or roentgenologic evidence of mediastinal or other localized masses exerting pressure on the nerve.

The visceral manifestations of vagal paralysis are exceedingly difficult to analyze. Some of our patients had bradycardia, others tachycardia. One has "vasovagal" attacks (?) with paroxysmal auricular fibrillation. In most, there were no disturbances of cardiac rhythm. Changes in respiratory rhythm or function did not occur in periods of freedom from laryngeal crises.

Kelly,[40] in experiments on dogs, has shown that, although both cortical and bulbar centers for the larynx exist, complete paralysis of the vocal cords cannot be obtained by cortical lesions alone; there must also be a lesion in the bulbar center. Destruction of the cortical centers does not impair respiratory movements of the cords. Disease in the medulla may be limited to one small area of the laryngeal centers, so that only one muscle is affected, e. g., the abductor. According to Kelly, the chief diseases of the medulla which, by involvement of the laryngeal centers, give rise to immobility of the vocal cords are tabes dorsalis, progressive bulbar paralysis,

39. Wilson, S. A. K., and Bruce, A. N.: Neurology, Baltimore, Williams & Wilkins Company, 1941, pp. 413-420.

40. Kelly, A. B.: Neurological and Mechanical Factors Underlying Immobility of the Vocal Cords: I. Neurological Aspect, Brit. M. J. **2**:678, 1927.

syphilitic arteritis and gummata, syringobulbia, disseminated sclerosis, hemorrhage, and softening from thrombosis and embolism (though only in tabes dorsalis does the typical laryngeal crisis occur). He has stated that laryngeal paralysis is encountered in 40% of the cases of tabes dorsalis.

REPORT OF CASES

The present-day rarity of clinically obvious laryngeal involvement in tabes dorsalis is exemplified by our own material. The files of the Syphilis Division of the Medical Clinic, The Johns Hopkins University School of Medicine and The Johns Hopkins Hospital, and of the private practice of one of us (J. E. M.) contain the records of approximately 1,500 patients with tabes dorsalis or taboparesis. Of these, only 11 [41] (about 0.6% have suffered from clinically apparent laryngeal paralyses or laryngeal crises. This is in striking contrast to the high incidence reported by Greene [29] (9 of 60 cases), Semon [36] (14 of 100 cases), Dorendorf [28] (28 of 235 cases), and Gräffner [30] (88 of 206 cases). It is true that in some of these older series the diagnosis was made on the basis of routine laryngologic discovery of a symptomless posticus paralysis which would otherwise have escaped attention. We, ourselves, have not as yet made a routine laryngologic study of a large series of tabetics, and our own incidence of 0.6%, in contrast to the 23% incidence of the four older series just cited, suggests either that most examples of laryngeal involvement in tabes dorsalis are symptomless (unilateral or even bilateral posticus paralysis) or that the incidence of such manifestations has greatly decreased in recent decades.

The report of our own cases follows.

CASE 1.—*Mild laryngeal crises (paroxysmal cough) with paresis, both abductor and adductor, of the right vocal cord in a young (37-year-old) woman with minimal tabes dorsalis; persistent tachycardia (rate 120 to 130) but no other evidence of vagal involvement; electrocardiography not done; infection congenital or accidentally acquired in childhood(?); first evidence of syphilis gumma of nasal septum in childhood; first evidence of tabes dorsalis (left internal ophthalmoplegia) at age 20; improvement and final disappearance of laryngeal symptoms after treatment.*

D. McM., a 37-year-old woman, a housewife, was first seen Dec. 17, 1947.

History of Syphilis.—This patient had been aware since early childhood—"as far back as I can remember"—of a large perforation of the cartilaginous portion of the nasal septum. Left internal ophthalmoplegia (greatly dilated left pupil, blurred vision), the etiologic background of which was not then recognized, developed when she was about 20. This persisted for some months and gradually disappeared.

In the history there was no definite suggestion of syphilitic infection in any member of the immediate family, including parents and five siblings, though efforts to examine them have been unsuccessful.

The patient herself married at the age of 28. Her husband, aged 37, is known to be nonsyphilitic. She had been pregnant only twice, resulting in healthy seronegative daughters, aged 8 and 3 years. The long duration of the patient's infection (congenital? acquired?) explains the normal status of husband and children.

Two months after the birth of the second child, three years previously (patient aged 34), she consulted a physician because of cough. For the first time a routine serologic test was performed, and it gave a positive reaction for syphilis. He had given her about two years of uninterrupted metal chemotherapy, during and after which she had gained 15 lb. (6.8 kg.)

41. There were actually 12 patients, including the patient in Langworthy's previously reported case.[38]

in weight (from 114 to 129 lb. [51.7 to 58.5 kg.]). Since the serologic reaction had remained positive, the patient became discouraged and requested consultation.

History of Laryngeal Difficulties.—The patient has had a chronic cough "mildly off and on" since babyhood. For 10 years, since she was 27, this cough had been becoming increasingly troublesome, so much so that another physician had ordered some five or six x-ray examinations of the chest during this period. He told her that she "had some spots on her lungs, but he didn't know what they were." Three years before her first visit to Baltimore the cough had become especially troublesome and had led to the discovery of the positive reaction to the serologic test.

The cough was always paroxysmal, "strangling in character," with inspiratory stridor, dry and nonproductive, and there had been no hemoptysis, night sweats, or loss of weight. Since the institution of antisyphilitic treatment by her family physician, it had improved somewhat but had not disappeared.

For 15 years or more (since about the age of 22) she had noted slight huskinesss of her voice, which was clearer at times than at others. There was no relation of this or the cough to upper respiratory infections.

History of Other Symptoms of Tabes Dorsalis.—The left ophthalmoplegia interna (its cause unrecognized at the time) had appeared first at the age of 20, some two years or more before any laryngeal symptoms. Other symptoms of tabes dorsalis, specifically inquired for, were disavowed.

General Examination (pertinent physical and laboratory data).—There were no definite stigmata of congenital syphilis, although both upper first molars were "mulberry" in type. There was a huge perforation of the cartilaginous nasal septum, which had been present since childhood. The nasopharynx was normal. The lungs were entirely clear, and roentgen examination of the chest showed minimal infiltration at the apex of the right lung suggestive of an old healed lesion of reinfection tuberculosis, the lung fields being otherwise clear. The findings on the remainder of the general examination and on routine laboratory examinations of blood and urine were normal except for an increased sedimentation rate (21 mm. corrected Westergren method).

Neurologic Examination: The pupils were normal in size, the left slightly larger than the right. Both were fixed to light but reacted normally in accommodation (the left internal ophthalmoplegia of 17 years previously had been replaced by the bilateral Argyll Robertson phenomenon). The right knee jerk was obtainable only on reinforcement, as were also the ankle jerks, though the left knee jerk was normally obtained. There were no other neurologic abnormalities.

Laryngologic Consultation (Dr. N. Floyd Adams Jr.): This disclosed that there was slight impairment of movement of the right vocal cord on both abduction and adduction, the left moving normally. Direct laryngoscopic examination was not performed. There was no evidence of infection or new growth in the nasopharynx or larynx. The relationship of this laryngeal palsy to tabes dorsalis was not appreciated by us until three years later, after experience with other cases; at the time it was dismissed as of unknown cause.

Laboratory Evidence of Syphilis: The blood serologic reaction for syphilis was positive with a quantitative titer of 32 dilution units. The spinal fluid contained no cells; there were 35 mg. of protein per 100 cc., and the complement fixation test gave a doubtful result with large amounts of fluid (0.6 and 0.8 cc.) and was positive with 0.1 cc.; the mastic curve was flat.

Further Course.—Since two years of metal chemotherapy had not provided complete relief from the cough, the patient was treated further, over a 10-day period, with 6,000,000 units of penicillin procaine in oil with aluminum monostearate. No further treatment was given. Shortly after this the cough and slight huskiness of the voice disappeared entirely and had not recurred three years later (February, 1951). The patient gained an additional 12 lb. (5.4 kg.) in weight (to 141 lb. [64 kg.]). There were now, however, further objective evidences of tabes dorsalis: the right knee and both ankle jerks were absent even on reinforcement. Deep pain sense in the Achilles tendons had been lost, and there was a narrow band of zonal hyperesthesia about the thorax at the level of the fifth and sixth thoracic vertebrae. In February, 1951, Dr. Proctor reported the larynx to be entirely normal. The laboratory findings were essentially unchanged.

CASE 2.—*Mild laryngeal crises followed by complete abductor paralysis of both vocal cords in a 41-year-old physician; tracheotomy necessary to prevent suffocation; syphilis of 19 years' duration; neurologic difficulties preceded by gumma of pharynx, later iritis; no other symptoms of tabes dorsalis; pupillary abnormalities and laboratory evidence only objective signs; complete recovery from symptoms of laryngeal palsy within six months; no further neurologic developments over 14 years; preataxic tabes dorsalis (?)*

(J. H. H. 6746) A 41-year-old white male physician, was admitted to the hospital July 21, 1934.

History of Syphilis.—At the age of 22, in 1915, a penile chancre developed which was followed in a few weeks by a generalized, including palmar and plantar, nonitching rash. On the basis of a diagnosis of primary and secondary syphilis, treatment was started, but it consisted only of 20 intramuscular injections of mercury over a two-month period.

At 26, in 1919, a gumma of the posterior pharyngeal wall developed. For this the patient received one course, totaling 5 gm., of neoarsphenamine.

At the age of 31, in 1924, he graduated from a South American medical school, and at this time, for reasons that are not clear, both blood and spinal fluid were examined and found to be "positive" for syphilis (no details). Because of this, he received two years of intermittent treatment with intravenous injections of neoarsphenamine and intramuscular injections of mercury, when treatment was again stopped without laboratory recheck. For the next eight years he received no further treatment.

In January, 1934, when he was 41, left iritis developed, because of which, although without further physical or laboratory examination, he received 4 further injections of arsenic and 20 of mercury over a 12-week period. The iritis improved rapidly.

History of Laryngeal Difficulties.—In January, 1934, at about the same time as the appearance of the iritis, the patient noticed "irritation, pain, and a burning sensation in the throat," together with a dry, nonproductive, paroxysmal cough. This was followed by some difficulty in breathing, and his voice became "soft" and hoarse. These symptoms gradually progressed in spite of the antisyphilitic treatment given for iritis.

On June 20, 1934, spasmodic choking and "complete obstruction of the throat" suddenly developed. Slight relief was obtained from morphine, but within a few hours tracheotomy (performed elsewhere) was necessary to prevent suffocation. The tube was still in place when he was admitted to the Johns Hopkins Hospital one month later.

History of Other Symptoms of Tabes Dorsalis.—None was elicited, in spite of direct questions.

General Examination.—The patient was of asthenic habitus and had recently lost weight. There was a large scar (of the old gumma) on the posterior pharyngeal wall. A tracheotomy tube was in place, and the lower portion of the incision was indurated and infected. The lungs and heart were normal on examination. Roentgen examination of the chest revealed only extensive fibroid infiltration in the upper lobe of each lung, interpreted as due to an old tuberculous infection. A mediastinal mass was not present. There was a scar on the dorsum of the prepuce. Results of the remainder of the examination were not significant. Routine laboratory examinations showed no abnormalities.

Neurologic Examination: This showed dilated unequal pupils, the left larger than the right. Both were noncircular, and both reacted only sluggishly and incompletely to light and in accommodation. The neurologic status was otherwise normal, and there were no demonstrable signs of posterior column damage.

Laryngologic Consultation (Dr. Julian Chisolm): This revealed bilateral abductor paralysis of the vocal cords. No other abnormalities were found.

Laboratory Evidence of Syphilis: The Kahn standard flocculation test of the blood gave a doubtful reaction; the serologic reaction with other techniques was negative. The spinal fluid contained 2 cells; the Pandy reaction was negative (quantitative protein estimation not done), the complement fixation reaction was positive with large amounts of fluid (0.8 and 1.0 cc.) and the mastic curve was flat.

Further Course.—Metal chemotherapy with arsenic and bismuth was continued (in South America) for an additional seven months, until February, 1935. A letter from the patient, received in 1948, reported that by December, 1934, it was possible for him to breathe comfortably

without the tube for short but increasingly long periods and that in January, 1935, the tube was permanently removed. Since then, breathing has been normal, and the hoarseness has entirely disappeared. No further laryngeal examinations were made.

His letter of 1948 reports no further symptoms of tabes dorsalis, and the results of "frequent neurologic examinations have been negative except for hyperactive knee jerks." A recheck of the serologic reaction of the blood for syphilis and of the spinal fluid has not been done.

Meanwhile he had suffered from and survived peptic ulcer, purulent sinusitis, "rheumatic spondylitis" with intervertebral ankylosis, acute pyelonephritis, and amebic colitis.

CASE 3.—*Complete paralysis of one vocal cord, and 20 months later of the other, in a 49-year-old white man in whom other definite symptoms and signs of tabes dorsalis developed simultaneously; tracheotomy; no recovery from laryngeal paralysis in the ensuing nine years; recently, attacks of unconsciousness—vagal attacks (?)*

(J. H. H. 247638) A 49-year-old white man was admitted to the hospital Dec. 9, 1941.

History of Syphilis.—There was no history of early infection. Six years previously (1935), having formerly been quite well, he became "terribly run down." His family physician found that a routine blood serologic test for syphilis elicited a positive reaction. Metal chemotherapy was begun, and the patient continued it irregularly and intermittently until his admission to The Johns Hopkins Hospital.

History of Laryngeal Difficulties.—In 1939, two years before admission, he began to suffer from paroxysmal attacks of choking, especially at night; his voice became hoarse, and he was found (elsewhere) to have a paralyzed vocal cord. In September, 1941, a particularly severe acute attack of choking developed; he was found at another hospital to have bilateral paralysis of the cords, and an emergency tracheotomy was done. The tracheotomy tube was still in place on his admission to the Johns Hopkins Hospital.

History of Other Symptoms of Tabes Dorsalis.—In 1939, concurrently with the appearance of the choking attacks, typical severe lightning pains in the legs developed. About five months before admission, slight ataxia, and urinary hesitancy and nocturnal incontinence appeared.

General Examination.—There was hypertension (190/120) and tachycardia (rate 120). The heart was not enlarged, and the sounds were clear except for a systolic aortic murmur. The cardiac rate did not change with position, carotid sinus pressure, or Valsalva's test.

Roentgen examination of the chest showed no abnormalities; an electrocardiogram was normal.

The remainder of the examination elicited essentially normal findings.

Neurologic Status: The patient had the classic signs of tabes dorsalis: miotic Argyll Robertson pupils, absence of ankle jerks, slight ataxia, diminished deep pain sense, relaxed anal sphincter, and neurogenic bladder.

Laryngologic Consultation (Dr. John Baylor, later Drs. Floyd Adams and Samuel Crowe): The voice was normal. The vocal cords were almost in the midline. Abduction was paralyzed on both sides and adduction was paralyzed on the left, but the right cord did adduct on phonation. The left cord was bowed. Dr. Proctor's interpretation of this is total recurrent laryngeal paralysis on the left and posticus paralysis on the right.

Laboratory Evidence of Syphilis: The blood serologic reaction for syphilis was weakly positive, with a quantitative titer of 2 units. The spinal fluid contained 30 cells, 25 mg. of protein per 100 cc., complement fixation was positive with 0.025 cc. of fluid, and the colloidal gold curve was 5555531000.

Further Course.—On Dec. 11, 1941, the patient was inoculated intravenously with tertian malaria. After about eight days of atypical and almost continuous fever (to 105 F.) he suffered shock, circulatory collapse, and transient auricular fibrillation, electrocardiographically confirmed. The malaria was terminated. This was later followed by 26 intravenous injections of tryparsamide, all treatment being discontinued in August, 1942.

He was observed at yearly intervals, last in November, 1948. There has been no improvement in the laryngeal paralysis, and continuous use of the tracheotomy tube is obligatory. All other symptoms of tabes dorsalis have, however, disappeared except for occasional bouts of lightning pains. The spinal fluid showed continuous improvement and by 1945 had become, and has remained, normal.

However, in May, 1947, he began to suffer from attacks of unconsciousness and in the next 18 months had six such episodes. Two weeks after the first one, he was shown (elsewhere) electrocardiographically to have had an "old or healing posterior myocardial infarction." The attacks of unconsciousness develop abruptly without warning and last 5 to 15 minutes. He usually vomits at least once while unconscious. In at least one such attack, he has elsewhere been shown electrocardiographically to have paroxysmal auricular tachycardia. In others, the pulse is said to be very rapid, and temporarily irregular in both force and rhythm. In at least one recent attack, he has had one or more generalized convulsive seizures (reported by mail in 1951).

CASE 4.—*Unilateral right vocal cord paralysis appearing abruptly in a 23-year-old white man with syphilis of three-and-a-half-years' duration; symptoms (but no physical signs other than laryngeal palsy) suggesting low-grade acute syphilitic meningitis (neurorecurrence?) appearing one to three months after lapse in irregular and inadequate metal chemotherapy; complete recovery within five months after further treatment, no significant neurologic developments seven years later*

(K. 79760) A 23-year-old white man was admitted to the hospital April 30, 1924.

History of Syphilis.—This young man had had repeated attacks of gonorrhea in the preceding five years. In 1921, three and a half years before admission, he had a penile sore, followed in a few weeks by a generalized skin rash, diagnosed as primary and secondary syphilis. In the ensuing three years, he had received irregular treatment with arsenic and mercury, the last in January, 1924. One month after this treatment stopped, he began to feel bad, with "jumping of nerves all over body," supraorbital headache, tinnitus, fatigability, tachycardia and palpitation, unsteadiness in gait, and loss of 15 lb. (6.8 kg.) of weight in spite of a voracious appetite.

History of Laryngeal Difficulties.—On April 27, 1924, three days before admission, his voice suddenly became very hoarse, without apparent cause. There was no history of an acute upper respiratory infection and no cough.

General Examination.—This revealed moderate generalized adenopathy. The heart was normal except for striking bradycardia. The cardiac rate in the recumbent position was 40 per minute, increased to 50 on sitting up, and only to 56 after vigorous exercise which brought out a marked sinus arrhythmia. The lungs were clear and a roentgen examination of the chest showed no abnormalities, with no evidence of a mediastinal mass. There were two scars on the corona glandis. Other abnormalities were lacking. Unfortunately, electrocardiography was not done.

Neurologic Examination: This showed nothing abnormal except markedly hyperactive deep reflexes in arms and legs. The eyegrounds and visual fields and findings on audiometer and vestibular tests were normal.

Laryngologic Consultation (Dr. Clyde Heatly): This disclosed a hoarse voice and a completely motionless right vocal cord (both abductor and adductor paralysis); the left vocal cord moved normally but not beyond the midline, producing a large triangular defect in the glottis on phonation. Dr. Proctor's impression was total right recurrent laryngeal paralysis.

Laboratory Data: The blood serologic reaction was repeatedly negative. The spinal fluid, however, contained 9 lymphocytes, complement fixation was positive with 0.6 cc. and larger amounts, and the mastic curve 2333000000.

Further Course.—From May 9, 1924 to September 18, 1925, the patient was treated irregularly with arsenic and mercury. The symptoms of headache and nervousness disappeared promptly. Unfortunately, no further observations of pulse rate are available. By Aug. 8, 1924, the voice was normal; the right cord now adducted slightly and the left moved past the midline. By Oct. 4, 1924, all evidence of laryngeal paralysis had disappeared. He was lost from subsequent observation.

CASE 5.—*Bilateral abductor paralysis of the vocal cords, with bradycardia and bradypnea, in a 27-year-old Negro man with syphilitic infection of unknown duration, and minimal signs of tabes dorsalis; outcome unknown*

(J. H. H. 189318) A Negro man aged 27 was admitted to the hospital Dec. 18, 1939.

History of Syphilis.—The patient had gonorrhea in 1932 at the age of 20, but he said that he had no lesions of early syphilis or antisyphilitic treatment. Five months before admission

intermittent sore throat and gradual weight loss (15 lb. [6.8 kg.]) developed. A few weeks later his family physician discovered a positive serologic reaction for syphilis, but no treatment had been given.

History of Laryngeal Difficulties.—One month before admission, the patient had paroxysmal spasms of choking, worse on lying down. About 10 days later, in one of these spells, he became apneic and cyanotic, and lapsed into unconsciousness. An emergency tracheotomy was performed at another hospital.

History of Other Symptoms of Tabes Dorsalis.—None were elicited on direct questioning.

General Examination.—There were bradycardia (rate 52) and bradypnea (rate 12). The chest, lungs, and heart were otherwise normal on physical examination, and roentgenograms of the chest showed no abnormalities. There was a small scar on the prepuce. Unfortunately, electrocardiography was not done.

Neurologic Examination: This examination showed slightly noncircular pupils, reacting sluggishly to light but normally in accommodation. The knee jerks were obtainable only on reinforcement; the ankle jerks were unobtainable. There was no other evidence of posterior column damage.

Laryngologic Consultation (Dr. Proctor): This disclosed bilateral abductor paralysis of the vocal cords; laryngeal expiration could be accomplished, but inspiration was impossible except through the tracheotomy tube.

Laboratory Data: The blood serologic reaction for syphilis was positive (not titered). The spinal fluid contained 10 cells, 25 mg. of protein per 100 cc., the results of the complement fixation test were positive with 0.6 cc. and larger amounts, and the mastic curve was 2210000000.

Further Course.—This was unknown, since the patient was lost from observation.

CASE 6.—*Taboparesis in a 54-year-old white man, first appearing 14 years after infection, 1925, and progressing, as to the tabetic component, until 1948, in spite of treatment; development of bilateral abductor paralysis of vocal cords in 1946; tracheotomy; partial recovery*

(N. Y. H. 81755) (Part of this man's history is reported through the courtesy of Dr. Bruce Webster): A white man, aged 54, was seen in consultation by one of us (J. E. M.) Nov. 4, 1946.

History of Syphilis.—The infection was acquired in 1911, but the early records have been lost and details are not available.

History of Laryngeal Difficulties.—In May, 1946, seven years after the first development of taboparesis the patient gradually became hoarse. On June 23, 1946, he suddenly experienced great difficulty in breathing ("suddenly I nearly choked to death"), and an emergency tracheotomy was done. The tube was still in place six months later.

History of Other Developments of Tabes Dorsalis.—In 1925, the patient had a brief attack of diplopia, clearing spontaneously in a few weeks. In 1933, severe lightning pains in the legs appeared. In 1934, he suffered a break of the right tibia and fibula (spontaneous fracture [?] not accompanied by pain) which was followed by osteomyelitis, necessitating amputation of the leg. Later hesitancy of urination and loss of libido and potentia developed. In 1940, and in spite of several years' treatment of various types, he experienced some mental difficulties, and his condition was diagnosed as taboparesis. Eventually, after further treatment, he recovered from the paretic component of the illness.

General Examination.—This examination (in 1946, six months after the laryngeal paralysis) disclosed peripheral neuritis of the remaining (left) leg, with foot drop. This was due presumably to alcohol, which he had been consuming in large amounts (about 2 qt. [about 2,000 cc.] daily of sherry, champagne, and brandy) for 18 months. There was moderate hypertension (blood pressure 174/108). A roentgen examination of the chest showed no abnormalities.

Neurologic Examination: This examination showed, as evidence of tabes dorsalis, unequal Argyll Robertson pupils; feeble right knee jerk (midcalf amputation on this side); absence of left knee and ankle jerk; zonal hypesthesia about the trunk; absence of deep pain sense in the testes, and relaxed anal sphincter.

Laryngologic Consultation (Dr. Clark Grove): The consultation in May, 1946, when hoarseness first developed, revealed "chronic laryngitis" and partial abductor paralysis of the

vocal cords. On June 23, with the sudden development of respiratory difficulty, the bilateral abductor paralysis had become complete.

Laboratory Data: In December, 1934, the blood serologic reaction for syphilis was positive. The spinal fluid cell count was increased (no details), the results of a complement fixation test were strongly positive, and the colloidal gold curve was 1123310000.

In June, 1946, at the time of development of the laryngeal paralysis, and after a great deal of antisyphilitic treatment, both the blood serologic reaction and the spinal fluid were normal.

Further Course.—Beginning in 1934 and ending about 1941, the patient received large amounts of metal chemotherapy, including at least 46 intravenous injections of arsphenamine, 5 Swift-Ellis treatments, 8 injections of tryparsamide (which resulted in toxic amblyopia with eventual spontaneous recovery from this), and 64 intramuscular injections of a bismuth salt. In addition, he was treated twice, in 1937 and again in 1941, with induced tertian malaria. No treatment was given between 1941 and 1946. As a result of this, he recovered from the paretic component of his illness, but his several tabetic symptoms (lightning pains, urinary hesitancy, loss of potentia) were not significantly improved.

When the laryngeal paralysis occurred in June, 1946, five years following, and in spite of, the above treatment, he was re-treated with 4,200,000 units of penicillin in 14 days. The hoarseness improved, and within a month or two he was able to close the tracheotomy tube for long periods without recurrence of respiratory difficulty. There was gradual return of vocal cord function, first on the left and later on the right. On Sept. 3, 1946, there was "slight limitation of abduction function bilaterally." In April, 1947, the tracheotomy tube was permanently sealed off, but the patient, fearful of a recurrence of his original attack, has refused to permit its removal.

As presumptive evidence that tabes dorsalis is still active, it should be noted that in January, 1948, a spontaneous fracture of the mandible developed.

On Feb. 14, 1951, the patient was reexamined laryngologically by Dr. James A. Moore, who reported "complete adduction of both vocal cords on phonation. There was approximately 70% abduction of the left cord and 50% abduction of the right cord on full inspiration. It appears that there has been little change in function since 1946, and there is still some limitation of abduction on each side."

CASE 7.—*Tabes dorsalis in a 42-year-old white man, manifested first by ataxia, then, a few months later, by bilateral abductor paralysis of the vocal cords; strangling and choking attacks on swallowing liquids; subjective but no objective improvement after two years of metal chemotherapy*

(U. 20750) A white man, aged 42, was admitted to the hospital Sept. 12, 1928.

History of Syphilis.—There was no history elicited.

History of Laryngeal Difficulties.—About 13 months before admission the patient gradually became hoarse and had intermittent "choking and strangling attacks." The latter occurred when he attempted to swallow liquids but not when he swallowed solid food. Simultaneously, he noted that inspiration tended to end in a crowing sound. There had been no cough or acute attacks of respiratory distress.

History of Other Evidence of Tabes Dorsalis.—A few months before the above developments, the patient noted that he was somewhat unsteady on his feet, especially in the dark.

General Examination.—There was moderate tachycardia (pulse rate 100) and hypertension (blood pressure 160/100). Roentgen examination of the chest showed no abnormalities, and a barium swallow under the fluoroscope indicated that the esophagus functioned normally. The strangling on liquids was so prominent a symptom as to cause the examining internist to insist on esophagoscopy, with negative results.

Neurologic Examination: This examination showed typical signs of tabes dorsalis. The pupils were noncircular and reacted very sluggishly to light. Knee and ankle jerks were not elicited and there was moderate ataxia of the legs.

Laryngologic Consultation (Dr. S. J. Crowe): Consultation established the existence of a normal voice at the time of examination and bilateral abductor paralysis of the vocal cords, with fixation of the arytenoids. Dr. Proctor later interpreted the history and these objective findings to indicate bilateral posticus paralysis and, in addition, bilateral superior laryngeal paralysis to account for the strangling on swallowing liquids.

Laboratory Data: The serologic reaction of the blood for syphilis was negative. The spinal fluid contained 10 cells per cu. mm.; the Pandy reaction was 3+; complement fixation, positive with 0.4 cc. and larger amounts; the mastic curve, 5543210000.

Further Course.—During the ensuing two years, the patient received uninterupted metal chemotherapy. This resulted in gradual improvement in, and eventual disappearance of, the strangling and choking on swallowing liquids, but objective examination of the larynx one year, and again two years, after admission showed no change in the laryngeal paralysis. During treatment other symptoms of tabes dorsalis, not previously present, appeared in the form of lightning pains in the legs and hesitancy in urination. The patient was then lost from observation.

CASE 8.—*Classic tabes dorsalis in a 37-year-old white man; early syphilis at 22; five years later extraocular muscle palsy; a year or so later gradually developing complete paralysis of the left vocal cord, persisting for 8 to 10 years; no laryngeal crises; subsequent appearance of gastric crises*

(C. 86030) A white man, aged 37, was admitted to the hospital Sept. 11, 1928.

History of Syphilis.—Fifteen years earlier, when the patient was 22, a penile sore developed; it was followed six weeks later by generalized mucocutaneous lesions. The blood serologic test for syphilis gave a positive reaction, and diagnosis of primary and secondary syphilis was made elsewhere. He was treated with mercury and potassium iodide for two or three years, and treatment was terminated at this time with a course of 6 to 8 weekly intravenous injections of arsphenamine.

History of Laryngologic Difficulties.—About 8 to 10 years before admission, and perhaps 5 or more years after infection, the patient gradually experienced a sensation of constant soreness in the throat—"it feels as if there were a growth on the right side of the neck, deep in." At the same time there began a gradual change in the timbre of his voice, which at first was mildly, then increasingly, husky and which, for two or three years, had assumed a high piping tone. There were no acute attacks of difficulty with respiration or phonation.

History of Other Evidence of Tabes Dorsalis.—A year or so before the first appearance of laryngeal symptoms, diplopia developed and for several months the patient's right eye turned outward. This condition disappeared spontaneously. About five years before admission, infrequent attacks of typical gastric crises with epigastric pain, nausea, and vomiting occurred. At about the same time, mild ataxia of the legs developed.

General Examination.—This examination gave essentially negative results. Roentgen examination of the chest showed no abnormalities.

Neurologic Examination: This revealed classic signs of tabes dorsalis, among them miotic Argyll Robertson pupils, absence of deep reflexes in the legs, and moderate ataxia.

Laryngologic Consultation (Dr. Stuart Egerton): The consultation disclosed complete abductor and adductor paralysis of the left vocal cord, which lay motionless in the cadaveric position. There was slight movement of the left arytenoid. The right cord moved normally on phonation and passed beyond the midline to approximate the motionless left cord. Dr. Proctor's interpretation of these findings was that the motion of the left arytenoid was undoubtedly attributable to the action of the right side of the interarytenoideus and that the patient had a total left recurrent laryngeal nerve paralysis, possibly combined with left superior laryngeal paralysis (because of the sensory disturbance).

Laboratory Data.—The serologic reaction of the blood for syphilis was negative. The spinal fluid contained 10 cells; the Pandy reaction was 2+; complement fixation, doubtful with 0.6 cc. but positive with 0.8 and 1.0 cc.; the mastic curve, 2210000000.

Further Course.—The patient was lost from observation.

CASE 9.—*Classic tabes dorsalis developed in a 49-year-old white man, 18 years after infection with syphilis; development of laryngeal crises without evidence of vocal cord paralysis during antisyphilitic treatment, uninfluenced by continuation of the treatment; eventual development of simple dilapidating paresis*

(G. 63608) A white man, aged 49, was admitted to the hospital April 29, 1922.

History of Syphilis.—At the age of 31, eighteen years before admission, the patient had a penile lesion which was treated locally only. No definite diagnosis had been made. There were no further immediate developments.

History of Tabes Dorsalis.—About a year before admission classic lightning pains in the patient's legs developed, and a few months later there were loss of libido and potentia, urinary hesitancy, and episodes of enuresis.

History of Laryngologic Difficulties.—Some six to eight months after admission, while undergoing uninterrupted metal chemotherapy, he suffered "choking attacks" which continued at irregular intervals for the next two years in spite of uninterrupted further treatment. The attacks were brief and were separated by periods of freedom ranging in length from several days to many months. On Aug. 1, 1924, about 27 months after admission, he had such an attack under direct observation in the clinic. Abruptly and without warning, he became intensely dyspneic and aphonic. There was marked inspiratory stridor with crowing at the end of inspiration, and he was unable to talk. He became moderately cyanosed but did not lose consciousness. Changes in cardiac rate and blood pressure were not observed. The entire attack lasted about five minutes and ended as abruptly as it had begun. Immediately at its termination, he was examined by a laryngologist.

General Examination.—The results of this examination were essentially negative. Roentgen examination of the chest showed no abnormalities.

Neurologic Examination: This examination revealed unequal miotic Argyll Robertson pupils. One observer noted that the uvula deviated slightly to the left. All deep reflexes in the extremities were absent. There was mild ataxia of the arms and more marked ataxia of legs.

Laryngologic Consultation (Dr. E. N. Broyles): Consultation was available within ten minutes of the cessation of the attack described above. There was no evidence of vocal cord paralysis, but the left cord jerked spasmodically during quiet respiration.

Laboratory data: On admission the blood serologic reaction was positive for syphilis. The spinal fluid contained 180 cells; the Pandy reaction was 4+, and complement fixation was positive with 0.1 cc. and larger amounts. The mastic curve was 5543200000.

Further Course.—The patient received uninterrupted metal chemotherapy for about three years. There was no improvement in the laryngeal crises. Mental deterioration gradually developed, and about 1927 he was committed to a state psychiatric hospital as insane due to paresis.

CASE 10.—*Tabes dorsalis in a 50-year-old white man; no history of infection; first manifestation lightning pains, followed three years later by spasmodic attacks of coughing ending in vomiting (like pertussis), persisting for years; two years later, gastric crises; situation complicated by huge retrosternal thyroid and multiple organic lesions of gastrointestinal tract; laryngeal attacks persisting for 15 years, but their tabetic nature unrecognized until 20 years after the original onset, when (1951) right posticus paralysis was recognized; laryngeal symptoms finally relieved, though objective paralysis persists*

(340973) A white man, aged 50, was admitted to the hospital Dec. 14, 1944.

History of Syphilis.—No history of infection or of early lesions was presented. He was first discovered to have a positive serologic reaction in 1933, some three years after the onset of the laryngeal difficulties described below. He then received three to five years of metal chemotherapy.

History of Laryngeal Difficulties.—About 1930 attacks of spasmodic paroxysms of coughing began which were preceded by a tickling sensation in the patient's throat and were so severe that they ended with regurgitation of food. He said, "I'll bet I have ruined 100 hats; I would get a coughing fit in a public place and would have to regurgitate immediately into the only thing available—my hat!" These attacks were first attributed to chronically infected tonsils, but removal of the tonsils gave no relief. The attacks gradually increased in frequency and severity, occurring daily, or nearly so. During the first three years of these attacks he lost 106 lb. (48.1 kg.) in weight, reducing from 243 (110.2 kg.) to 137 lb. (62.1 kg.).

Most of the attacks of paroxysmal coughing ended in the immediate regurgitation of food, as in the paroxysmal cough of pertussis, but with gradually increasing frequency some of the attacks ended in periods of prolonged and intractable vomiting which lasted for days or weeks and were not associated with epigastric pain. These terminated as abruptly as they began, though in the course of some of them the patient became seriously dehydrated and required hospitalization. He was examined by many physicians because of these attacks, and numerous diagnoses

were made, including duodenal diverticulum, gall-bladder disease, and myocardial infarction, none of which was ever confirmed.

After three years of this the blood serologic test reaction for syphilis was discovered to be positive and antisyphilitic treatment was instituted, as outlined above. This treatment effected immediate improvement. Within three weeks the attacks described ceased more or less abruptly and, instead of occurring daily, occurred very infrequently, only once or twice a year. The patient began to gain weight and in about a year had regained 80 lb. (36.3 kg.).

The patient remarked that prior to the onset of this illness he had been a professional singer but that after the onset he had never been able to sing without precipitating a coughing attack.

History of Other Manifestations of Tabes Dorsalis.—About 1930, three years before the onset of laryngeal trouble, characteristic lightning pains developed. About 1935, the laryngeal crises began to be accompanied by, or followed by, gastric crises, likewise described above, and sometimes the gastric crises occurred without any precipitation by a laryngeal crisis. He had lost libido and potentia to some degree, beginning about 1941.

General Physical Examination.—Examination disclosed a large nontoxic adenoma of the thyroid, which had been present for 10 to 12 years. The trachea was deviated to the left. There was widening of the retromanubrial dullness, moderate tachycardia, and blood pressure of 146/90. Roentgenologic examination of the chest showed that the thyroid was very much enlarged and extended into the chest to the level of the top of the arch of the aorta. Other roentgenologic examinations were noncontributory.

Neurologic Examination: This examination revealed unequal pupils, the left fixed to light, the right reacting sluggishly, and both reacting normally in accommodation. The right knee jerk was normally active, but the left was only sluggishly obtained, and both ankle jerks were absent. There was some loss of deep pain sense in the Achilles tendons and the testes and markedly delayed pain sense in the soles. The anal sphincter was greatly relaxed.

Laryngological Examination: This was unfortunately not carried out at the time of the patient's original admission, since the coughing attacks were, as it now appears later, erroneously attributed to pressure on the trachea from the greatly enlarged substernal thyroid. In 1951, however, viewing the patient in restrospect from the standpoint of possible laryngeal involvement of tabes dorsalis, laryngoscopic examination was carried out by Dr. Proctor who reported that the right arytenoid and cord appeared to be relatively motionless and in the midline. Dr. Proctor believed that the patient had a right posticus paralysis, and it seems certain from the course of events that this had been present since about 1930.

Laboratory Data: The patient's spinal fluid is reported to have been first examined in 1933 and is said to have been normal. In the ensuing years and prior to admission to the Johns Hopkins Hospital his spinal fluid was retested on four or five occasions and was always reported normal. At the time of his admission here the blood serologic test reaction for syphilis was positive, with a quantitative titer of 2 units, and has remained so since. His spinal fluid has been examined by us on two occasions and has been normal on both, except for the presence of a moderate increase in precipitation in the first three tubes of the mastic test.

Further Course.—The patient has had a stormy and complicated career. A retrosternal thyroid about as large as a child's head was removed by Dr. W. M. Firor in January, 1945. The operation did not produce any further damage to the recurrent laryngeal nerves. This proved to be a nontoxic thyroid adenoma. In the ensuing six years, however, the retrosternal thyroid, probably incompletely removed, has recurred, and there is once again evidence of some pressure on the trachea and esophagus. In these years, also, he has been demonstrated to have a considerable number of complicating disorders in the gastrointestinal tract, including a traction diverticulum of the middle third of the esophagus, duodenal ulcer, duodenal diverticulum, multiple diverticuli of the large bowel, gallstones, and internal and external hemorrhoids. He has had multiple bronchopulmonary infections. In March, 1950, a large gall bladder, containing a large solitary stone about the size of an olive, was removed, as was also an enormous but otherwise normal appendix about 18 cm. long. In the meanwhile, however, he has been almost completely relieved of the symptoms of laryngeal difficulties, has had no spontaneous laryngeal crises, and is not now and never had been hoarse, the only change in his voice being, as indicated above, that he can no longer sing without precipitating a cough.

Other symptoms of tabes dorsalis have continued. He has occasional bouts of severe lightning pains and infrequent attacks of gastric crises.

CASE 11.—*A white man, aged 45, admitted February, 1939, with taboparesis; no history of early syphilis; first neurologic symptom spasmodic coughing attacks in 1932, followed in 1937 by urinary hesitancy, lightning pains, paresthesias, ataxia, weight loss, and mental symptoms of simple deterioration, later manic-grandiose psychosis; complete recovery from paretic component of illness and marked improvement from most tabetic symptoms after malaria—metal chemotherapy maintained for 12 years; no improvement in laryngeal symptoms; in 1951 questionable right posticus paralysis, with roentgenologic evidence of neurogenic involvement of upper third of esophagus*

(J. H. H. 163355) A 45-year-old white man was admitted to the hospital Feb. 28, 1939, complaining of the after-effects of an injection of a bismuth salt (given elsewhere) into the left sciatic nerve.

History of Syphilis.—There was no history of syphilis given. The patient was first discovered to have a positive serologic reaction for syphilis in the course of routine examination early in 1938, some five years after the first onset of neuropsychiatric (especially laryngeal) symptoms; metal chemotherapy was begun shortly afterward and continued until his admission to the Johns Hopkins Hospital.

History of Taboparesis.—Laryngeal symptoms first appeared in 1933. About 1937, urinary hesitancy, urgency, frequency, and occasional incontinence developed, but libido and potentia were retained. Coincident with this were occasional bouts of typical lightning pains in the legs, numbness of the feet, slight ataxia, gradual loss of 22 lb. (10 kg.) in weight, and mental symptoms. The last were loss of business efficiency, increasing periods of unwarranted irritability, tenseness, and mild depression.

History of Laryngeal Difficulties.—These were so overshadowed by other and more serious manifestations of taboparesis that little attention was paid to them, and their nature was unrecognized until 1951, fourteen years after his original admission to the Johns Hopkins Hospital. At that time (1951), the following history was elicited.

About 1932, he began to suffer with attacks of spasmodic cough, unrelated to any local irritation. These attacks were precipitated by talking in a loud voice, especially if he shouted or attempted to sing, by talking too long, thus tiring his voice, and sometimes by swallowing, especially if he ate or drank too rapidly. He illustrated one phase of this by remarking that in his capacity as an official of the Masonic order he was frequently called upon to read a long passage of the ritual, requiring 30 minutes for completion. He learned by experience that in the middle of this he had to break off for about a minute and drink a glass of water lest he precipitate a spasm of intractable and unproductive cough. The attacks appeared at irregular intervals, sometimes several a day, sometimes with periods of freedom for a week or more. He had never been hoarse.

In August, 1932, early in the course of this symptom, he suffered a violent coughing and choking attack while eating a pickle and was thought to have inhaled a portion of it. He was referred to Dr. Louis Clerf, of Philadelphia. As the patient recalled it 19 years later, Dr. Clerf told him that there was something wrong with the muscles in the back part of his throat. Upon inquiry in 1951, Dr. Clerf reported that the complete record had been lost but that the results of a bronchoscopic examination were negative as were also roentgen studies of the chest. Dr. Clerf stated, "I have no record of anything wrong with the ligaments around his vocal cords."

The patient has had slight to moderate enlargement of the thyroid gland (simple nontoxic goiter) in the neck for many years, and from time to time during our observation of him it has been thought that his coughing attacks might be due to tracheal compression from this or from a retrosternal thyroid. In pursuit of this idea, he was referred to Dr. J. W. Pierson in 1945 and again in 1947 for roentgen examination. There was no evidence of retrosternal thyroid or of compression of trachea or esophagus. However, Dr. Pierson noted on both occasions that there was difficulty in initiating the act of swallowing, with temporary ballooning of the first portion of the esophagus. Once the barium entered the esophagus, it passed through normally. Without knowledge of the patient's general condition, Dr. Pierson offered the

brilliant suggestion that this abnormality was probably on a neurogenic basis. As for ourselves, the import of Dr. Pierson's suggestion was not appreciated until several years later.

General Physical Examination.—In 1939, this examination revealed undernutrition with marked weight loss, a soft systolic murmur in the aortic area, blood pressure of 144/80, and slight to moderate diffuse enlargement of the thyroid gland. A roentgenogram of the chest disclosed no abnormalities.

Neuropsychiatric Examination: The pupils were large, equal, and circular, and reacted only sluggishly and slightly to light but normally in accommodation. The knee and ankle jerks were not elicited. There was moderate ataxia of the lower extremities and loss of position sense of the great toes, relaxed anal sphincter, diminished pain sense to pinprick over the fronts of both shins, and absence of pain sense in the soles.

From the psychiatric standpoint, there were slight defects in memory, calculation, and retention. The patient was overtalkative and rambling in conversation. There were tremor of the facial muscles and moderate typical paretic dysarthria. Insight was lacking.

Laboratory Data.—The serologic reaction of the blood for syphilis was positive with a titer of 33 units. The cerebrospinal fluid contained 11 cells, and 40 mg. of protein per 100 cc. The Wassermann reaction was positive with 0.1 cc. and larger amounts, and the mastic curve was in the first zone.

Treatment and Subsequent Course.—In March, 1939, the patient was treated with induced tertian malaria, in the course of which an excited manic-grandiose psychosis developed, necessitating the patient's temporary psychiatric hospitalization for two months. After this episode, he had a complete remission from the paretic component of his illness, which has been maintained for the ensuing 12 years.

Postmalarial metal chemotherapy was given for about a year, ending in July, 1940, since which time no further treatment has been given.

Of his tabetic symptoms, the urinary troubles and the lightning pains have wholly disappeared and the ataxia is greatly improved. Objectively, no additional evidence of neurologic damage has appeared. From the laboratory standpoint, the cerebrospinal fluid, examined at yearly intervals, has gradually reverted to complete normality, and the blood serologic titer has decreased to 1 unit. On the other hand, there has been no change whatever in the laryngeal attacks, which have now persisted for nearly 20 years. The patient has learned to minimize them by avoiding the several precipitating factors enumerated above.

Laryngologic Examination: Since the nature of the laryngeal attacks was not recognized until 1951, there is, unfortunately, no record of any laryngoscopic examination until that date. In July, 1951, Dr. C. C. Douglass reported: "I first thought there was limitation of motion of the right vocal cord, but repeated examination showed definite movement throughout the normal range. The vocal cords themselves were essentially normal. The larynx is normal except for possible periodic restriction of motion."

COMMENT

An analysis based only on clinical data, without pathologic observations, in the small number of 11 cases, is subject to obvious limitations. Nevertheless, something of present-day interest may be gained.

Justification for Tabes as Etiologic Factor in Laryngeal Paralyses and Crises.—This rests in part on the older literature in which, as indicated above, the association has been repeatedly pointed out and in part on the exclusion of other apparent causes. In all our cases, sources of extrinsic pressure on the vagus in the neck or its recurrent laryngeal branch in the neck were sought, both by clinical examination and by roentgen study of the chest. None was found, except the retrosternal thyroid in Case 10, and in this case the earlier and subsequent course of events makes it reasonably certain that the laryngeal symptoms were due to tabes dorsalis rather than to the thoracic mass.

TABLE 4.—*Summary of Clinical Features of*

Case	Race	Sex	Age	Duration Syphilis on Admittance, Years	Admission Diagnosis	Symptoms of Tabes Dorsalis Antedating Laryngeal Difficulties	Objective Evidence of Tabes Dorsalis (Other than Laryngeal)	CSF* Group	Laryngeal Symptoms	Duration Laryngeal Symptoms Before Correct Etiologic Diagnosis	Laryngeal Crisis
1	W	F	37	30+	Tabes dorsalis	Left ophthalmoplegia interna at age 29; no other symptoms	Minimal	2	Paroxysmal "strangling" cough; inspiratory stridor; slight hoarseness since age 27	13 yr., of which 3 yr. under personal observation	+
2	W	M	41	19	Preataxic tabes dorsalis (?)	None	Pupillary changes only; no evidence posterior column damage	2	Laryngeal paresthesias, cough, hoarseness for 6 mo. in 1934, followed by abrupt onset spasmodic choking and inability to inspire	7 mo.	+
3	W	M	49	6 (?)	Tabes dorsalis	Lightning pains concurrent with laryngeal symptoms	Classic	3	Paroxysmal choking, hoarseness; one vocal cord paralyzed 2 yr.; sudden onset respiratory obstruction	2 yr.	+
4	W	M	23	3½	Preataxic tabes dorsalis (?) meningeal neurosyphilis (?)	None	None	2	Sudden hoarseness 3 days' duration	3 days	0
5	N	M	27	?	Tabes dorsalis	None	Minimal	2	Paroxysmal choking 2 weeks, then sudden complete respiratory obstruction with apnea, cyanosis, coma	1 mo.	+
6	W	M	54	35	Taboparesis	1925 diplopia; 1933 lightning pains; 1934 painless spontaneous fracture right leg; 1936 neurogenic bladder, impotence; 1940 paretic psychosis	Classic	3	1946 hoarseness, followed by sudden attack choking, respiratory obstruction	1 mo.	0
7	W	M	42	?	Tabes dorsalis	Slight ataxia 6 mo. before laryngeal symptoms	Classic	3	Increasing hoarseness; choking and strangling attacks	13 mo.	+
8	W	M	37	15	Tabes dorsalis	Diplopia 1 yr. earlier	Classic	2	About 7 yr. after infection, gradual onset of laryngeal soreness and hoarseness	8 yr. +	0
9	W	M	49	18	Tabes dorsalis	Lightning pains, impotence, neurogenic bladder 1 yr. before	Classic	3	Choking attacks, spasmodic dyspnea, and aphonia	2 yr.	+
10	W	M	50	?	Tabes dorsalis	Lightning pains 3 yr. earlier	Classic	Neg.	Paroxysmal cough and vomiting, like pertussis	20 yr., of which 6 yr. under personal observation	+
11	W	M	45	?	Taboparesis	None	Classic	3	1933 paroxysmal spasmodic cough	18 yr., of which 12 yr. under personal observation	+

* CSF = cerebrospinal fluid.

Laryngeal Involvement in Tabes Dorsalis

Laryngeal Paralyses	Tracheotomy necessary.	Other Symptoms Vagus Paralysis	Associated Cranial Nerve Paralysis	Chest Roentgenogram	Subsequent Course (After Treatment) of	
					Laryngeal Symptoms	Other Evidence Tabes Dorsalis
Partial abductor and adductor paralysis right vocal cord (recurrent laryngeal); left normal	0	Tachycardia	Partial of left 3d cranial (ophthalmoplegia interna)	Normal	Partial improvement after 2 yr. of metal chemotherapy; complete disappearance of symptoms 2 mo. after penicillin; paralysis absent 3 yr. later	Further objective (but not subjective) manifestations of tabes dorsalis have developed since cessation of treatment 3 yr. ago
Bilateral abductor paralysis, complete, (recurrent laryngeal); questionable superior laryngeal involvement (paresthesias)	+	None	0	Normal	Complete recovery in 6 mo.; no recurrence in subsequent 14 yr.	None (report by mail from physician-patient)
Complete abductor and adductor paralysis left; abductor paralysis right (recurrent laryngeal)	+	Tachycardia; later development of "vasovagal" (?) fainting attacks and convulsive seizures, paroxysmal auricular tachycardia	0	Normal	No improvement 10 yr. later; "vasovagal" (?) attacks increased in frequency and severity	No further developments
Complete abductor and adductor paralysis right cord (recurrent laryngeal); left normal	0	Bradycardia (rate 40 per minute)	0	Normal	Complete recovery 5 mo.; lost from further observation	Lost from observation
Bilateral abductor paralysis (recurrent laryngeal)	+	Bradycardia (rate 52); bradypnea (rate 12)	0	Normal	No data	Lost from observation
Bilateral abductor paralysis (recurrent laryngeal)	+	None	Diplopia with recovery 21 yr. before laryngeal symptoms	Normal	Partial recovery 6 to 8 mo. permitting sealing tracheotomy but partial abductor palsy persisting 5 yr. later	No improvement tabetic symptoms; spontaneous fracture of mandible, 1948
Bilateral abductor paralysis (recurrent laryngeal); questionable superior laryngeal (strangling)	0	Tachycardia	0	Normal	Crises disappeared after 2 yr. metal chemotherapy; but palsy persisted 4 yr. then lost from observation	Progression; lightning pains and neurogenic bladder
Complete abductor and adductor paralysis left (recurrent laryngeal); possible left superior laryngeal	0	None	Diplopia with spontaneous recovery	Normal	No data	Lost from observation
None, but spasmodic jerking, left cord	0	Uvula deviated to left (?) (pharyngeal branch?); otherwise none	0	Normal	No improvement in 5 yr.; then lost from observation	Continued paretic mental deterioration
Posticus paralysis right (recurrent laryngeal)	0	Tachycardia	0	Retrosternal thyroid (see text)	Improvement after 3 wk. metal chemotherapy; but infrequent attacks persist for 20 yr., as does partial posticus paralysis	Progression; gastric crises developed and persist
1951 questionable weakness right posticus	0	Weakness upper ⅓ esophageal musculature (see text)	0	Normal	Infrequent attacks persist for 18 yr.	Progression 4 yr. after onset laryngeal symptoms; various other manifestations tabes dorsalis and paretic psychosis developed, later relieved by appropriate treatment

Incidence of Laryngeal Troubles in Tabes Dorsalis.—As already pointed out, and as accepted at present, these are rare manifestations of tabes dorsalis. We have been able to find only 11 symptomatic cases in a careful review of the cases of some 1,500 tabetic patients. The clinical symptoms are so strikingly obvious when present that their existence could hardly have failed to be noted, though, as our own experience shows, they are not infrequently misinterpreted. Because of our own clinical obtuseness, it is probable therefore that a few more cases do exist in our own material which were not detected in our survey.

We have no explanation for the relatively great frequency of laryngeal symptoms of tabes dorsalis in the latter half of the last century, as reported in the older literature, and their comparative rarity now. Further, and for lack of routine laryngoscopic examination in a large series of tabetics without laryngeal symptoms, we have no data as to the present-day occurrence of silent posticus paralysis.

Accuracy of Diagnosis.—As the introductory paragraph to this paper points out, the laryngeal manifestations of tabes dorsalis were apparently familiar enough to our medical predecessors. They have been so little stressed in the literature of the past 30 years that most internists, neurologists, and laryngologists are totally unfamiliar with them. This is well exemplified by the fact that in our own material laryngeal symptoms had been present for more than one year in 7 of our 11 patients (for 8, 13, 18, and 20 years, respectively, in 4 patients) before their relationship to tabes dorsalis was recognized. We, ourselves, failed to appreciate the situation in three of these patients during 3, 6, and 12 years, respectively, of personal observation, and we made the correct diagnosis for these three patients only after interest in the present clinical study had been aroused (especially by Case 3, the first of our own cases to be properly identified).

Race and Sex Distribution.—In our 11 cases, 10 of the patients are males, and only 1 is female; 10 are white and only 1 is a Negro. Even though the number of our cases is small, the preponderance of white males is substantially greater than in our tabetic patients as a whole.

"Preataxic" Development of Laryngeal Manifestations.—The older authors, especially Fournier,[6] lay great stress on the observation that laryngeal crises and paralysis are usually "preataxic," i. e., the first obvious clinical manifestations of tabes dorsalis. This is not our experience. Seven of our 11 patients had definite, sometimes severe, clinical evidence of tabes dorsalis antedating any laryngeal difficulties. In two the laryngeal symptoms were definitely "preataxic," being followed later by other tabetic signs or symptoms (Cases 5 and 11). In two others (Cases 2 and 4), the diagnosis of tabes dorsalis is questionable. In Case 2 the patient showed only pupillary abnormalities as clinical evidence of neurosyphilis (other than the laryngeal paralysis) and is reported by letter to have manifested no other signs or symptoms in the ensuing 14 years. For this patient the correct diagnosis may be diffuse meningovascular neurosyphilis. The patient in Case 4 had had syphilis only three and a half years at the time of the development of the laryngeal paralysis, and he presented no other signs or symptoms of neurosyphilis. The patient has been lost from later observation, but his case may represent an example of low-grade syphilitic meningitis [42] rather than tabes dorsalis.

42. We have seen several examples of recurrent laryngeal nerve paralysis as a part of clear-cut early acute syphilitic meningitis, both as a part of early syphilis in the absence of treatment and as neurorelapse. These cases are not considered in this article.

Laryngeal Crisis Versus Laryngeal Paralysis.—The older literature suggests that (*a*) laryngeal crises often occur in the absence of laryngeal paralyses, and (*b*) of patients with vocal cord paralysis, only about half have crises. In our own material, the first of these observations is not borne out. We have seen only one patient (Case 9) with crises in the absence of paralysis and in him there was observed the "laryngeal ataxia" of the older writers, i. e., spasmodic jerking of a single vocal cord. The remaining 10 patients all had one or another type of laryngeal paralysis, and of these, 7 also had laryngeal crises.

Clinical Aspects and Severity.—The symptoms described by our patients conform so exactly to those reported by Fournier [6] and others that a review of them is not necessary. It need only be pointed out that in 4 of our 11 patients tracheotomy was essential as a lifesaving measure because of suffocating attacks produced by bilateral abductor paralysis. We have observed no cases in which the condition was fatal.

Clinical Localization of the Lesion.—In all of our cases, so far as can be judged from inadequate data (inadequate in part because of insufficient knowledge of the anatomy and physiology of the vagus nerve, in part because the several consulting laryngologists were not at the time aware of the neurophysiologic implications of the lesion), the recurrent laryngeal nerve was involved. This was bilateral and partial in four cases (abductor paralysis only), bilateral and complete on the left and partial on the right in one, unilateral (complete on the right) in two, unilateral (left complete) in one patient, and right partial in three patients. The tendency toward left-sided paralysis noted in the older literature does not appear.

Previous papers make little reference to superior laryngeal (sensory) paralysis in this situation. Its existence was suspected by one of us (D. P.) in three of our cases (*2*, *7*, and 8) on the basis of symptoms, but an objective sensory examination of the area supplied by this nerve was not performed in any of our cases.

Purely on clinical grounds, our material suggests that the lesion is peripheral rather than central and that, unlike most of the fiber tract degenerations in tabes dorsalis, the motor fibers of the recurrent laryngeal rather than the sensory fibers of the vagus are usually attacked. The meager necropsy evidence suggests, however, that the lesion is actually central, involving the vagal nuclei.

Associated Symptoms of Vagus Nerve Paralysis.—Here our own observations are wholly inadequate, as are also (so far as we are aware) all other reports. Of our 11 patients, 4 had noteworthy tachycardia and 2 others had bradycardia (of these, 1 also had bradypnea). One patient (Case 3) has had remarkable "vasovagal" attacks, characterized by fainting, convulsive movements, and paroxysmal auricular tachycardia but has not been properly studied during an attack. One patient (Case 9) may have had some involvement of the pharyngeal branch of the vagus, but this rests on a single (and questionable) observation that the uvula deviated to the left. One remarkable patient (Case 11) had roentgenologically demonstrable weakness of the musculature of the upper third of the esophagus, and, indeed, it was this roentgenologic observation which finally led (18 years late!) to the recognition of the tabetic origin of his paroxysmal spasmodic cough.

It is regrettable that in our patients appropriate roentgenologic examination of the upper gastrointestinal tract and electrocardiographic and pharmacodynamic

studies of vagus function (as well as objective sensory examination) of the larynx have not been carried out. The omission will be remedied as opportunity presents itself.

Associated Cranial Nerve Lesions of Neurosyphilis.—It is notable that in eight of our cases, the vagus was the only cranial nerve involved. In one (Case 1) the laryngeal crises and paralysis had been preceded seven years earlier by partial third nerve paralysis (ophthalmoplegia interna) on the opposite side and in two others by diplopia (observed elsewhere and the exact lesion not defined) 1 year and 21 years earlier, respectively. In no instance here reported was there simultaneous involvement of another cranial nerve, though in Langworthy's case [38] the 9th and 11th nerves were also affected. This fact is also mildly in favor of peripheral rather than central vagal involvement.

Prognosis of Laryngeal Crises and Paralyses.—Of the eight patients with crises (with or without paralysis), four were completely relieved (by treatment?) within a few months to two years; two were partially relieved, but infrequent crises persist after 18 and 20 years, respectively, and one patient has remained unimproved after 10 years. In one case information is lacking.

Of the nine patients with objective paralyses of the vocal cords, three had complete clearing, three had partial clearing (some objective paralysis persisting after 2, 5, and 20 years, respectively), and one showed no improvement after 10 years; two were lost from observation. Viewed as a whole, laryngeal difficulties disappeared completely in only three of our nine patients on whom data are available and persisted in the remaining six for observation periods ranging from 4 to 20 years. This is in sharp contrast to Fournier's opinion [6] that spontaneous recovery is the rule.

Further Progression of Other Signs and Symptoms of Tabes Dorsalis.—Long-term data are lacking on three of our patients. Of the remaining eight, six showed further progression of other objective or subjective manifestations of tabes dorsalis (or paresis) in spite of prolonged treatment. This observation suggests (but, owing to the limited number of available cases, far from proves) that the development of laryngeal involvement in tabes dorsalis indicates a relatively poor prognosis for the future general course of the disease.

SUMMARY AND CONCLUSIONS

1. We have reviewed the literature on laryngeal involvement in tabes dorsalis and report 11 cases of our own.

2. The symptoms produced by laryngeal crises and paralyses are described. Crises may occur with or without paralysis, and vice versa.

On the basis of the older literature, the commonest form of paralysis is completely asymptomatic unilateral posticus paralysis, discoverable only on routine laryngoscopic examination.

Symptoms may range from relatively mild and infrequent attacks of paroxysmal spasmodic unproductive cough to severe apoplectiform attacks of choking, suffocation, apnea, convulsive seizures, coma, and death (in sudden bilateral abductor paralysis of the vocal cords). Tracheotomy may be necessary as a lifesaving measure.

3. Laryngeal involvement in tabes dorsalis has been relatively rare in recent years (in our material occurring in perhaps 1 to 3% of a group of some 1,500

tabetic patients). It appears to have been much more frequent in the latter half of the 19th century. The explanation for the discrepancy is not clear, but perhaps it lies in the usual omission of routine laryngoscopic examination of tabetic patients, with consequent failure to detect asymptomatic posticus paralysis.

4. The comparative rarity of the lesion and the lack of recent publications on the subject in the medical literature are responsible for the unfamiliarity of most physicians with the clinical syndrome, and for a usual long delay in correct diagnosis.

5. Our limited material suggests that laryngeal involvement is commonest among white male tabetics and much less frequent in Negroes and in females.

6. Contrary to statements in the older literature, laryngeal involvement in tabes dorsalis is not usually "preataxic," i. e., the first obvious clinical manifestation. It is usually preceded by other symptoms and is accompanied by classic physical signs of posterior column damage.

7. On clinical grounds alone, the literature and our own cases suggest that the pathologic lesion is probably peripheral in the vagus nerve, usually the recurrent laryngeal branch, rather than central, though scanty pathologic evidence (in four cases) suggests the opposite.

The extent to which the superior laryngeal (censory) branch or the visceral functions of the vagus may be involved has never been adequately studied, and our own cases do not supply the omission.

8. Again contrary to the opinions expressed in the older literature, the prognosis of laryngeal involvement in tabes dorsalis is poor. Laryngeal signs and/or symptoms disappeared in only three of our nine patients who could be followed, persisting in the other six for periods ranging from 4 to 20 years. It is also suggested that laryngeal involvement is an unfavorable prognostic omen for the general course of tabes dorsalis, which in these cases is usually progressive in spite of treatment.

803-807 Medical Arts Building.

Case Reports

BLASTOMYCOSIS OF LARYNX

LIEUTENANT COMMANDER NELSON HORA OLIVEIRA (MC), BRAZILIAN NAVY

CASES of cutaneous and visceral blastomycosis are frequently seen and even cases of primary laryngeal forms are not rare. The present case seems worthy of publication, however, because of the unusual location of the lesion involving the vocal cord only, without any other presenting lesions. Very few case reports of this nature have been found in the literature. Even in cases of lesions involving the mouth or pharynx and reaching the larynx, in advanced stages only do the lesions reach the vocal cords.

REPORT OF A CASE

A white man, aged 46, a stewards mate in the Brazilian Navy, stationed in the Naval Tuberculosis Sanatorium in Friburgo, came to the otolaryngology clinic of the Central Naval Hospital on Oct. 15, 1947, complaining of hoarseness of two months' duration. Otolaryngologic examination disclosed the presence of a pale left vocal cord, rather infiltrated, with small ulcerations and granulations involving the posterior two-thirds. Mobility was somewhat impaired. A general examination, including x-rays of the lungs and blood tests, showed essentially normal conditions. A biopsy of the affected cord was performed on Oct. 23, and specimens were sent to two histopathologists. Each returned a diagnosis of blastomycosis. The patient was immediately placed on sulfathiazole therapy with a daily dose of 9 gm. administered for 20 days. The laryngeal lesions immediately began to subside under this therapy. On the 10th day of treatment the vocal cord was almost normal, and on the 15th day no sign of disease could be noted. Therapy was very well tolerated as indicated by urine and blood tests. At the present time, three years later, the patient appears to be in perfect condition, and there is every reason to consider his lesion completely healed.

COMMENT

The classification of blastomycosis remained in a confused state until 1938, when F. Nino proposed a classification which brought some clarity to the subject. According to Prof. F. E. Rabello, who made a slight modification on Nino's classification, blastomycosis can be divided into the following groups:

Group I: Genuine blastomycosis, such as the Busse-Buschke type

Group II: Parablastomycosis, including Gilchrist and Lutz's mycosis

Group III: Pseudoblastomycosis, including the Coccidioides infection of Posadas-Dickson

Group IV: Blastomycotic histiocytomatosis, such as Darling's malignant and J. Lôbo's benign diseases

The second group, parablastomycosis, is the most important to us, since it contains the neotropical form which is commonest in Brazil. This form was very thoroughly studied by Lutz in 1908 and received its present name from that investigator. Sulfonamide therapy for blastomycosis was first reported in Brazil by Oliveira Ribeiro in 1940 and may be said to have established a new era in the treatment of blastomycosis. Prior to the introduction of the sulfonamides blastomycosis was a very serious disease. Today it is almost benign. The second group

is said to contain the form most sensitive to the sulfonamides. It is interesting to note that in 1939 Prof. R. D. Sanson for the first time successfully treated a patient with blastomycosis of a vocal cord by electrocoagulation through a laryngofissure.

Physicians in Brazil do not generally favor the high doses of sulfonamides which are advocated in the United States, where a blood concentration of 15 mg. per 100 cc. and more is commonly reached. Here a concentration of 3 to 5 mg. per 100 cc. is, in general, sufficient. However, our patient received a greater amount of the drug, since facilities for checking the blood concentration were not available.

SUMMARY

A report of a case of blastomycosis of the larynx cured by sulfonamide therapy is presented. A brief survey of classification of blastomycosis is presented together with some considerations regarding its treatment with sulfonamide drugs. The chief interest of this case lies in the localization of the lesion on the vocal cord only.

Avenida Copacabana, 7, Apartamento 802, Rio de Janeiro.

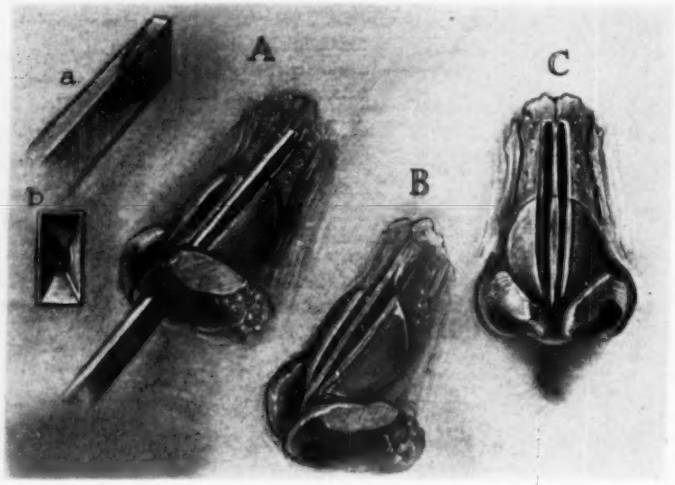
Clinical Notes, New Instruments and Techniques

SQUARE EXCAVATOR FOR USE IN NARROWING NASAL BRIDGE

A. P. SELTZER, M.D., Sc.D.
PHILADELPHIA

All surgeons who operate on the nose realize from experience that the best results and the fewest complications are present when skill in operating includes careful avoidance of unnecessary tissue injury in all steps of the procedure.

Surgical success has grown steadily with improvement in methods of operating, and such methods have, in turn, depended upon the devising of the instruments especially designed for the need to meet satisfactorily the various requirements of the different technical processes. Individual



Side and end view of square excavator (*a* and *b*); method of use and results (*A*, *B*, and *C*).

methods of operating are recognized, and for their carrying out there is the personal choice of a special type of instrument for each purpose.

For my own use, I have worked out a new model for an excavator for removing fragments of nasal bones, especially when this is done in the course of an operation for removal of a dorsal hump.

In the course of the classic surgical technique for removal of a hump, after the primary incision has been made, the perichondrium and periosteum have been elevated, and the excess of dorsal cartilage and bone have been removed, the dorsum of the bridge is too wide and flat and requires narrowing. This is done by making the maxillary processes movable, so that their upper margins may be pushed medially and approximated. To do this satisfactorily, more bony tissue is removed from the nasal bones in the subglabellar region.

My model of an excavator has been especially designed for this purpose. It consists of a rectangular steel rod 6½ in. (16.5 cm.) in length by ¼ by 1/16 in. (6 by 1.5 mm.). In one end, a box-like cavity is hollowed out, providing a rectangular cutting edge (Fig., *a* and *b*). It has been prepared in three sizes to suit the dimensions of the individual operative field.

To use this excavator, one inserts it through the primary incisions and passes it inward, so that it lies along the septum (Fig., *a*). When contact is made with the lower margin of the bone from which a piece is to be removed, the excavator is driven backward to the desired point by means of light taps with a mallet. This technique frees the bone segment from the nasal bone except at the narrow posterior extremity. The excavator is then withdrawn, and the narrow piece of bone which has been separated is twisted free with hemostatic forceps and removed (Fig., *B* and *C*). The same process is then repeated on the other side. If there are any rough edges remaining, they are smoothed down with a curved curette. The maxillary processes can now be pressed inward sufficiently to form a suitable nasal contour.

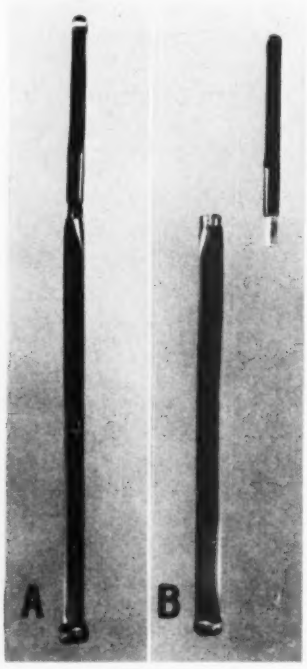
I have found this instrument to be more efficient than other models in general use, since it removes more easily and exactly the desired amount of bone from the bridge in narrowing this region after a hump has been excised.

2104 Spruce St.

"THROW-AWAY" BUTTON-END KNIFE BLADE

IRVING B. GOLDMAN, M.D.
NEW YORK

The button-end knife commonly used in rhinoplasty has to be extremely sharp because of its required precision. Owing to sterilization, cutting of cartilage, and completion of hump-severance, it becomes dull very readily. Thus the button-end knife requires almost constant sharpening and frequent replacement.



A, throw-away blade in permanent handle. *B*, blade disengaged by turning screw at bottom of permanent handle.

For many years I have tried to devise an interchangeable button-end knife similar in principle to the Bard-Parker handles and blades. I have now constructed one in which the button-end blade can be inserted, fixed, and changed as often as desired. The handle, which is perfectly balanced, has a screw at the bottom, which on being turned and pushed forward disengages and releases the blade. A fresh blade can be re-engaged by reversing the action of the screw.

Instrument manufactured by A. Klenk Instrument Co., Maspeth, N. Y.

From the Division of Rhinoplasty, Department of Otolaryngology, Mount Sinai Hospital.

Presented as a New Instrument at the Fifty-Sixth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 17, 1951.

The new handle with the "throw-away" button-end knife blade has the added advantage of economy.

It is not unlikely that in the future other blades, so easily dulled in rhinoplastic surgery, will be of the same "throw-away" type on similar handles, so that they can be economically and readily changed.

121 E. 60th St.

MODIFICATION OF HARTMANN FORCEPS

EPHRAIM L. MANNING, M.D.
DAVENPORT, IOWA

It was felt that it would be of value to have a forceps which could grip cerumen or other semisoft material without biting through it and tearing it, especially for removing cerumen which did not come out of the ear canal easily by irrigation. The Hartmann forceps (Fig. 1) and other types which come to a point at the end usually tear through the material or, having sharp edges, cut through it.

Fig. 1.—Hartmann forceps.

Realizing how easily the ear canal can be traumatized, I had preferred the blunt Buck ring curettes for removing cerumen. It was felt that if two small blunt rings could be combined in a forceps, the rings with blunt sides could give a wide surface to grip without having sharp edges which might cut.

An instrument like this, consisting of a Hartmann type forceps with jaws consisting of two blunt rings, each 2 mm. in diameter, was made (Fig. 2).[1]

At first, the instrument did not seem to do the work for which it had been constructed because it cut through the cerumen it grasped. However, it was soon realized that if one grasped some semisoft material and did not bring the rings tightly together, that is, kept them 1 or 2 mm. apart, the instrument was very useful and one could remove lumps of cerumen without just tearing off small pieces.

1. The instrument was made up by the Laidlaw Surgical Instrument Co., 627 E. Garvey Ave., Garvey, Calif.

The forceps was found to get an effective grip on aural polyps. It is inadvisable to exert force on an aural polyp because the polyp may be attached to ossicles or the wall of the labyrinth and one can injure these in pulling the polyp out. The forceps, however, helps hold the polyp while a snare is manipulated around it.

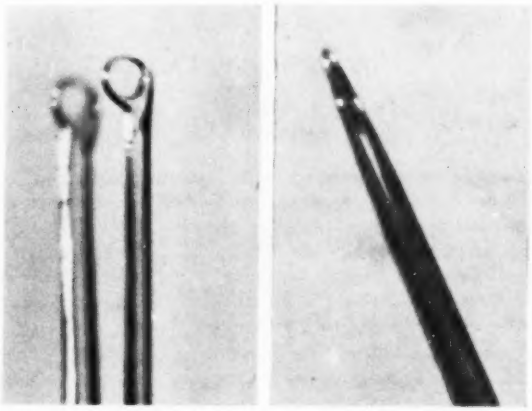


Fig. 2.—Manning modification of Hartmann forceps.

In removing a nasal polyp with a long stalk which had slipped down into the nasopharynx, the modified forceps was found useful to lift the polyp back into the nose, especially since its small bulk would fit into very small spaces in the nose and still permit one to see around it. For the same reason, it has been used on nasal and aural foreign bodies.

1820 W. 3rd St.

INTRABRONCHIAL CALIPER

ERNEST B. EMERSON Jr., M.D.
ROCHESTER, N. Y.

The need for accurate quantitative reports of bronchoscopic findings has become increasingly important with the development in recent years of thoracic surgery. A technique for obtaining linear measurements was described at an earlier date by me.[1] The thoracic surgeon, however, also wants to know whether a partially stenotic bronchus is present and, if so, the size of the lumen that remains. From the thoracic surgeon's point of view, a report to the effect that a bronchus is one-third or one-half closed is not satisfactory. If no more accurate reports can be obtained, he is often forced to do his own bronchoscopy.

To make such quantitative reports of the bronchial lumen possible, the intrabronchial caliper has been developed. The instrument allows the endoscopist to obtain quickly and easily the necessary measurements in the main bronchi with a minimum of trauma to tissue which may already have become friable with disease.

Fig. 1.—Intrabronchial caliper. The clock gives size of the lumen directly in French sizes.

INSTRUMENT

The instrument (Fig. 1) actually is an adaptation of a Riba urethrotome. It is 55 cm. in length from the forward tip to the attachment of the recording dial and slightly over 3 mm. in diameter. Through the barrel of this instrument runs a piece of German silver wire. It is attached at the peripheral end, and when pressure is applied via the clock and handle the wire bulges through a slit in the side of the end-piece.

The clock, at the proximal end of the instrument, automatically records the degree of bulge in the silver wire at the far end and reports it in French sizes. It was decided to use French sizes, as most of the problems involved after endoscopy have to do with tubes and catheters for which this measurement is most convenient. Conversion to millimeters in diameter is a matter of simple arithmetic.

TECHNIQUE

The technique of intrabronchial luminal measurement is easy, and the diagrammatic drawing (Fig. 2) will be self-explanatory to the endoscopist. The instrument is merely passed through the bronchoscope to the depth indicated and the handle on the dial turned until the examiner sees that the expansion of the wire below has reached the maximum possible at the site in question. Actually, after a little practice with the instrument, the operator can tell when the end-point has been reached by the sensation of resistance transmitted to his fingers from the dial handle.

From the Department of Surgery, Division of Oto-Rhino-Laryngology, University of Rochester School of Medicine and Dentistry, and in conjunction with the Bronchoscopic Services of the Veterans Administration Hospitals at Batavia and Canandaigua, N. Y.

1. Emerson, E. B.: A Bronchial Yardstick, J. Thoracic Surg. **21**:516-518 (May) 1951.

SUMMARY

An instrument, termed an intrabronchial caliper, for obtaining luminal measurements within the main bronchi has been described. The instrument has been found satisfactory as to ease and safety in actual use. The accuracy of the measurements obtained are satisfactory to the thoracic surgeon.

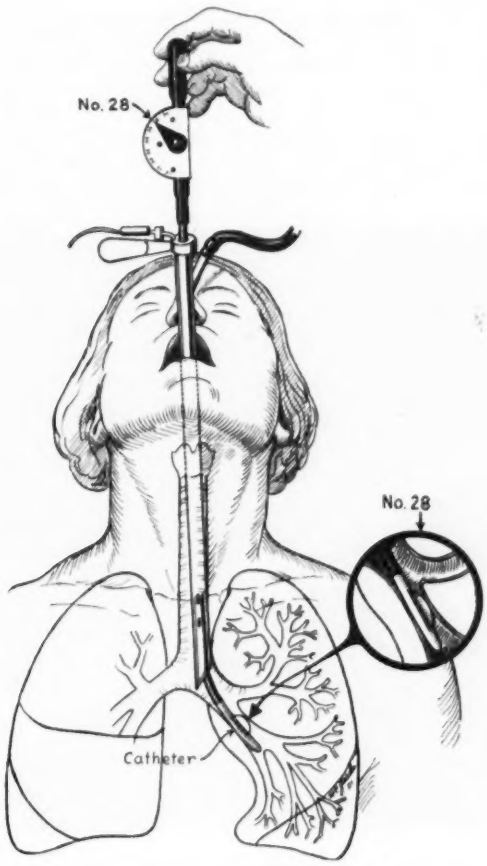


Fig. 2.—Diagrammatic drawing of caliper in use. The size of the bronchus measured is 28F., as shown in the insert and recorded on the dial.

It is hoped that the introduction of this instrument along with its predecessor [1] will further the development of quantitative bronchoscopic reporting.

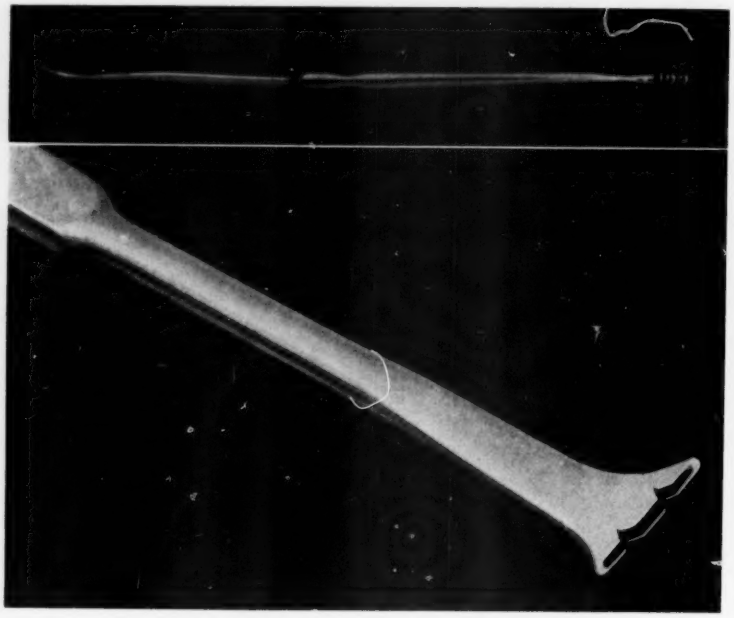
The intrabronchial caliper may be obtained from C. R. Bard, Inc., 490 Morris Ave., Summit, N. J.

26 Strathallan Park (7).

ASPIRATING TONSIL DISSECTOR

OTTO C. HIRST, M.D.
PHILADELPHIA

While performing a dissection tonsillectomy, one sometimes encounters hemorrhage, particularly at the supratonsillar fossa. This occasionally is so profuse that suture is required for its control before further dissection of the tonsil and its enucleation are possible.



Aspirating tonsil dissector

In a technique used in which the capsule of the tonsil is stripped from the pharyngeal aponeurosis and the superior constrictors of the pharynx, the instrument I am presenting is most useful in holding the aponeurosis and muscle while the tonsillar capsule is stripped from it. It is useful not only from this standpoint but also from the standpoint of any further dissection of scar tissue with a saw-like movement of the serrated edge of the instrument and aspiration of blood from the operative field at the same time.

The instrument is 20 cm. long. One end is flattened, notched, and tubular to permit suction of fluids. This flattened end is 18 mm. wide, permitting the instrument to be used as a light suction tongue depressor. It is very useful in dissection of the lingual tonsil.

From the Otolaryngological Service "B," Episcopal Hospital.

Read before the College of Physicians of the Section of Otolaryngology, Philadelphia, Feb. 6, 1951.

George P. Pilling & Son Company finished the instrument and the photographs for slides in July, 1950, after about three years' work.

Dr. Shinji Yoshida, of Kyushu, Japan, published an article in the March, 1950 *Annals of Otology, Rhinology and Laryngology,* describing a somewhat similar instrument on which he had worked 11 years. From a review of Dr. Yoshida's article and the pictures of his instrument, it is my impression that my instrument is the easier of the two to use for the technique previously mentioned because the dissecting end is wider. This width of the spatulous end would also more thoroughly clean the field of blood and saliva and less easily puncture the pharyngeal mucosa. The notches of the serrated edge of my instrument are deeper and the distance between the notches wider than in the Yoshida instrument. There is, therefore, less chance of the instrument slipping from the field of operation. It is my impression that the Yoshida instrument has a very small suction aperture and that my instrument is more easily cleaned and will not clog as readily as the Yoshida instrument.

Dr. Yoshida states that his instrument can be used not only for tonsillectomies but also for other operations in the ear, nose, and throat. I have tried my instrument only in the pharynx, but I believe that it can be used in other parts of the body.

3601 Stokley St.

Special Reports

HEARING AIDS ACCEPTED BY THE COUNCIL ON PHYSICAL MEDICINE AND REHABILITATION OF THE AMERICAN MEDICAL ASSOCIATION

As of June 1, 1952

Audicon Models 400 and 415

Mfr., National Earphone Co., Inc.
20 Shipman St.
Newark 2, N. J.

Audivox Model Super 67

Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Aurex Models L and M

Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Beltone Symphonette Model

Beltone Mono-Pac Model M

Mfr., Beltone Hearing Aid Company
1450 W. 19th St.
Chicago 8

Cleartone Model 500

Cleartone Model 700

Cleartone Regency Model

Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Dahlberg Model D-1

Dahlberg Junior Model D-2

Mfr., The Dahlberg Company
2730 W. Lake St.
Minneapolis 16

Dysonic Model 1

Mfr., Dynamic Hearing Aids, Inc.
149 Church St.
New York 7

Electroear Model C

Mfr., American Earphone Co., Inc.
10 E. 43rd St.
New York 17

Gem Model V-35

Gem Model V-60

Mfr., The Gem Ear Phone Co., Inc.
50 W. 29th St.
New York 1

Maico UE Atomeer

Maico Quiet Ear Models G and H

Maico Model J

Mfr., The Maico Company, Inc.
21 N. 3rd St.
Minneapolis 1

Mears (Crystal and Magnetic) Aurophone Model 200

1947-Mears Aurophone Model 98

Mfr., Mears Radio Hearing Device Corp
1 W. 34th St.
New York 1

Micronic Model 303

Micronic Model "Mercury"

Micronic Star Model

Audivox, Inc.—Successor to Western electric Hearing Aid Division.
123 Worcester St.
Boston 18

Microtone Audiomatic T-5

Microtone Classic Model T-9

Microtone Model T-10

Microtone Model T612

Microtone Model 45

Mfr., The Microtone Company
Ford Parkway on the Mississippi
St. Paul 1

National Cub Model (C)

National Model D (Duplex)

National Standard Model (T)

National Star Model (S)

National Ultrathin Model 504

National Vanity Model 506

Mfr., National Hearing Aid Laboratories
815 S. Hill St.
Los Angeles 14

Otarion Model E-4

Otarion Models F-1, F-2, and F-3

Otarion Model G-2 & G-3

Mfr., Otarion, Inc.
159 N. Dearborn St.
Chicago 1

Paravox Model D "Top-Twin-Tone"

Paravox Model J (Tiny-Myte)

Paravox Model XTS ('Xtra-Thin)

Paravox Model Y (YM, YC, and YC-7) (Veri-Small)

Mfr., Paravox, Inc.
2056 E. 4th St.
Cleveland 15

Radioear Permo-Magnetic (Multipower)

Radioear Permo-Magnetic (Uniphone)

Radioear All-Magnetic Model 55

Radioear Model 62 Starlet

Radioear Model 72

Mfr., E. A Myers & Sons, Inc.
306 Beverly Rd.
Mt. Lebanon
Pittsburgh 16
Distr., Radioear Corporation

Rochester Model R-1

Rochester Model R-2

Mfr., Rochester Acoustical Laboratories, Inc.
117 Fourth St., S. W.
Rochester, Minn.

Silvertone Model J-92

Silvertone Model P-15

Mfr., Johnston Hearing Aid Mfg. Co.
708 W. 40th St.
Minneapolis 8
Distr., Sears, Roebuck & Company
925 S. Homan Ave.
Chicago 7

Solo-Pak Model 99

Mfr., Solo-Pak Electronics Corp.
Linden St.
Reading, Mass.

Sonotone Model 700

Sonotone Model 900

Sonotone Models 910 and 920

Sonotone Model 925

Sonotone Model 940

Sonotone Model 966

Mfr., Sonotone Corporation
Elmsford, N. Y.

Super-Fonic Hearing Aid

Mfr., American Sound Products, Inc.
1303 S. Michigan Ave.
Chicago 5

Televox Model E

Mfr., Televox Mfg. Company
1307 Sansom St.
Philadelphia 7

Telex Model 22

Telex Model 97

Telex Model 99

Telex Model 200

Telex Model 300-B

Telex Model 400

Telex Model 1700

Mfr., Telex, Inc.
Telex Park
St. Paul 1

Tonamic Model 50

Mfr., Tonamic, Inc.
12 Russell St.
Everett 49, Mass.

Tonemaster Model Royal

Tonemaster Cameo Model

Mfr., Tonemaster, Inc.
400 S. Washington St.
Peoria 2, Ill.

Unex Midget Model 95

Unex Midget Model 110

Unex Models 200 and 230

Mfr., Nichols & Clark
Hathorne, Mass.

Vacolite Models J and J-2

Mfr., Vacolite Company
3003 N. Henderson St.
Dallas 6, Texas

Western Electric Models 65 and 66

Mfr., Audivox, Inc.—Successor to Western Electric Hearing Aid Division
123 Worcester St.
Boston 18

Zenith Model 75

Zenith Miniature 75

Zenith Model Royal

Zenith Model Super-Royal

Mfr., Zenith Radio Corporation
5801 W. Dickens Ave.
Chicago 39

(All the accepted hearing devices have vacuum tubes.)

Accepted hearing aids more than five years old have been omitted from this list for brevity.

TABLE HEARING AIDS

Aurex (Semi-Portable)

Mfr., Aurex Corporation
1117 N. Franklin St.
Chicago 10

Precision Table Hearing Aid

Mfr., Precision Hearing Aids
5157 W. Grand Ave.
Chicago 39

Sonotone Professional Table Set Model 50

Mfr., Sonotone Corporation
Elmsford, N. Y.

Abstracts from Current Literature

Ear

Proper Use of Antibiotics in Treatment of Acute Otitis Media. Miriam H. Rutherford, California Med. **75**:98 (Aug.) 1951.

The problem of preventing loss of hearing following acute otitis media has been made more complex by the use of penicillin and other antibiotic agents which may apparently cure yet leave dangerous residual disease. The causes of loss of hearing must be recognized early if remedial treatment is to be effective. In children, particularly, loss of hearing may go unnoticed for some time. Physicians who treat otitis media should feel the responsibility not only for bringing an acutely ill child back to health but also for preserving the function of the hearing mechanism. Careful examination of the ear after apparent subsidence of infection is mandatory. It is of the utmost importance to be able to recognize the tympanum in its normal state and its various pathological states and to be alert to the early signs of changes associated with loss of hearing.

Pryor, Redwood City, Calif. [A. M. A. Am. J. Dis. Child.].

Analysis of 500 Cases of Hearing Disability. Hayes A. Newby and Dorothy Munz Molyneaux, J. Speech & Hearing Disorders **16**:213 (Sept.) 1951.

Two hundred and ninety-two females ranging from 6 to 89 years, with a median of 45 years, and 208 males ranging from 6 to 81 years, with a median of 46 years, were classified under 10 types of hearing loss and the classifications compared with referral diagnoses of nerve deafness, otosclerosis, and mixed deafness. Nerve deafness may be diagnosed on a basis of an increasing loss from frequency to frequency in both bone and air conduction and with relatively poor speech sound discrimination. Diagnosis of otosclerosis may be based on a flat or slightly rising pure-tone audiometric pattern, relatively normal bone conduction, and relatively good speech sound discrimination.

Palmer, Wichita, Kan.

Evaluation of Hearing Aids Worn by School Children. Carl H. Weaver, J. Speech & Hearing Disorders **16**:218 (Sept.) 1951.

An evaluation of hearing aids worn by 20 school children, ranging from the 4th to the 10th grade, indicates (1) that the amount of gain in speech reception afforded by the aid apparently has little relation to the amount of time the aid is worn each day; (2) that there is little connection between hearing loss, both aided and unaided, and the amount of time the aid is worn each day; (3) that at these ages girls wear their aids significantly more than boys; and (4) that the amount of residual hearing seems to have no relation to the length of time the hearing aid has been worn each day.

Palmer, Wichita, Kan.

Survey of Attitudes of Local Industry Toward Employability of Hearing Aid Wearer. Alice J. van Deventer and Joseph A. Scanlon, J. Speech & Hearing Disorders **16**:222 (Sept.) 1951.

A survey of the attitudes of local industries by means of 476 returned questionnaires from the Aural Rehabilitation Clinic of the United States Naval Hospital in Philadelphia shows (1) that business is favorable toward hiring a man who wears a hearing aid but unfavorable toward hiring a man who has a hearing loss and will not wear such an aid; (2) that a man wearing a hearing aid is more acceptable for employment than men with other disabilities; (3) that insurance is not an important item in accepting or rejecting an applicant; (4) that ability and personality are still considered important items in determining employability, despite the

wearing of a hearing aid, and (5) that business believes in the selective placement of the hearing-aid wearer.

PALMER, Wichita, Kan.

CONGENITAL AURICULAR FISTULA. BENJAMIN BOFENKAMP, Minnesota Med. **34**:329 (April) 1951.

Congenital auricular fistula has been explained as an inclusion of epidermis in one of the grooves existing between the six tubercles forming the external ear. The condition may be bilateral or unilateral. When the tract becomes infected, it may be mistaken for a sebaceous cyst. Permanent cure can be obtained by surgical removal following injection of methylene blue.

STOESSER, Minneapolis [A. M. A. AM. J. DIS. CHILD.].

EFFECTS OF LIMITED HEARING ON THE DEVELOPMENT OF SPEECH IN CHILDREN UNDER 3 YEARS OF AGE. H. C. HUIZING and DOREEN POLLACK, Pediatrics **8**:53 (July) 1951.

Natural development of speech elements in acoustically handicapped children has been proved possible provided that the limited hearing is detected at a very early age by new testing procedures. It is achieved under clinical conditions by the use of individual hearing aids which provide auditory stimulation from the age of 2 years, or even earlier, upward. Special treatment is necessary, and training of the parents plays an important part. If a listening function is not established at an early age, there is a danger that children with limited hearing will ignore sound or will actually find it annoying. Future success is dependent upon the hard of hearing child's being educated together with normal-speaking children, with the reservation that he will need special arrangements for speech therapy and speech reading. New specifications should be made concerning the admission of acoustically handicapped children into special schools, and the considerations mentioned above should be applied.

FROM THE AUTHORS' SUMMARY [A. M. A. AM. J. DIS. CHILD.].

INCREASED FREQUENCY OF AUDITORY LESIONS ON COMBINED INTRASPINAL AND INTRAMUSCULAR ADMINISTRATION OF DIHYDROSTREPTOMYCIN. MARIANNE KAHL, Deutsche med. Wchnschr. **76**:944 (July 20) 1951.

Although the number of observations is somewhat limited, it is concluded that combined intraspinal and intramuscular administration of dihydrostreptomycin is followed as frequently by severe damage of the acoustic nerve as is that of streptomycin. It seems probable that dihydrostreptomycin is even more toxic. The observations were made on patients of the University Children's Clinic at Frankfurt, Germany.

AMBERG, Rochester, Minn. [A. M. A. AM. J. DIS. CHILD.].

CHARACTERISTICS OF ELECTRICAL COCHLEAR RESPONSE UNDER INFLUENCE OF VARIOUS CONDITIONS APPLIED TO CONDUCTIVE SYSTEM OF MIDDLE EAR. J. UKIDA, J. Oto-Rhino-Laryng. Soc. Japan **55**:8 (Jan.) 1952.

The author studied the change of response intensity as influenced by the experimental interference applied to the conductive systems of the middle ears of cats and guinea pigs.

1. Incision of the tympanic membrane produced a fall of response voltage of not more than 20 db.

2. Circumcision of tympanic membrane caused a voltage loss of 25 to 35 db., or more, and a shift of stimulus sound pressure by 30 to 60 db. in all frequencies. Removal of malleus or incus entailed a more marked loss.

3. Mercury instillation in the attic produced a loss of 2.5 to 5.0 db. in all frequencies. When the middle ear cavity was filled with mercury, the response fell in all frequencies, especially in the high frequencies above 2048 cps.

4. In a case of chronic otitis media the response was uniformly decreased by 14 to 23 db. over the whole range of frequency.

HARA, Los Angeles.

STUDIES ON "FOWLER PHENOMENON": CHAPTER II. STUDIES ON THE "ALTERNATE BINAURAL LOUDNESS BALANCE TEST" WITH REGARD TO OBSTRUCTION AND NERVE DEAFNESS. Y. SHIOTANI, J. Oto-Rhino-Laryng. Soc. Japan **55**:14 (Jan.) 1952.

The author applied the "alternate binaural loudness balance test" in 77 cases of obstructive deafness and 25 cases of nerve deafness, using a 49-A Audiometer.

Recruitment of loudness occurred only in cases of nerve deafness with unequal threshold of bone conduction between the two ears, while it did not appear in obstruction or nerve deafness with equal threshold of bone conduction between the two ears.

From this result, the author concludes that the "alternate binaural loudness balance test" is very important for differential diagnosis between nerve deafness and obstructive deafness.

HARA, Los Angeles.

Pharynx

INTRAVENOUS PROCAINE FOLLOWING TONSILLECTOMY. KENNETH SOMERS, Ann. Otol. Rhin. & Laryng. **60**:175 (March) 1951.

Inasmuch as good results have been reported when a dilute solution of procaine is given intravenously for the treatment of a variety of traumatic and painful conditions, Somers utilized this preparation in an attempt to relieve post-tonsillectomy pain. A discussion of the mode of action and probable safe therapeutic intravenous dosage of procaine is presented.

Forty-one patients were given intravenous injections of procaine hydrochloride on the day following tonsillectomy. The first group, of 18 patients, received 10 cc. of a 1% solution, while the second group, of 20 patients, received 10 cc. of a 2% solution and a third group, of 3 patients, were given 20 cc. of a 2% solution. All injections were administered within a five-minute period. In the first group, analgesia of the tonsil fossae lasted for an average of two hours, while patients in the second group experienced analgesia for about four hours and those in the third group for nine and one-half hours. A fairly high blood level of procaine was demonstrated in five of the patients 15 minutes after the intravenous injection.

Unpleasant symptoms experienced by patients during injection are believed to preclude the possibility that this form of therapy will become of value in routine post-tonsillectomy care.

STEELE, Kansas City, Kan.

TONSILLECTOMY AND POLIOMYELITIS: II. STUDIES ON INCIDENCE IN 1949. MORRIS SIEGEL, MORRIS GREENBERG, and M. C. MAGEE, J. Pediat. **38**:537 (May) 1951.

From a detailed statistical study of 53,007 tonsillectomies performed in New York in 1949, the authors conclude that tonsillectomy predisposes to the development of clinical symptoms of poliomyelitis. Fifty cases of poliomyelitis were reported from this group. Most of the patients were under 10 years old. The most hazardous months for operation are April, May, and June, particularly June; the safest months for tonsillectomy in New York appear to be December, January, February, and March. MACDONALD, Pittsburgh [A. M. A. AM. J. DIS. CHILD.].

THE ARTERIAL TENSION IN THE COURSE OF TONSILLECTOMY UNDER LOCAL ANESTHESIA. J. P. DEREYNIER, Practica oto-rhino-laryng. **12**:333, 1951.

DeReynier performed a tonsillectomy on a young woman, using a local anesthetic of 1% procaine hydrochloride with epinephrine. There were no immediate after-effects, but one-half hour after the completion of the operation the patient suffered a complete collapse, became unconscious, and was in a true state of shock. Under proper stimulation she made a recovery in about two hours.

Being impressed by this episode and recalling the work of Escher on fluctuations in arterial tension as a late aftermath of tonsillectomy, the author made detailed observations of the blood pressure both before and after operation of 33 patients who had undergone tonsillectomy under local anesthesia. Ten of his patients showed a slight elevation, of 20 to 40 mm. Hg, after the anesthetic; 10 other patients evidenced a marked hypertension, while in 13 there was a slight drop in the tension immediately following the actual surgery. However, beginning with the first

half-hour after the completion of the surgery there was a decided drop in the tension. In 22 cases the tension fell from 5 to 50 mm. Hg below the normal. In these cases the author feels that the delayed reactions of the patient are due not to the procaine but rather to the epinephrine. This is particularly noticeable in emotional and anxious patients or those who have a neurovegetative dystonia, those with a very labile arterial tension, and particularly those who have a tendency toward hypotension. He suggests that all local surgery should be done in a well established hospital, that an evaluation of the patients be made, and if they seem to fall into any of the above-mentioned groups, that one should be prepared for any possible late reactions. Proper preoperative medication should be instituted, epinephrine eliminated from the solution if possible, or, better still, the operation performed with the patient under general anesthetic.

PERSKY, Philadelphia.

CLINICAL OBSERVATION OF MALIGNANT TUMORS OF THE ORAL CAVITY AND PHARYNX. I. NAKAGAWA, J. Oto-Rhino-Laryng. Soc. Japan **55**:46 (Jan.) 1952.

Nakagawa reviewed the case records of 2,247 patients who were seen in the radiological therapy department of the Japanese Cancer Hospital from Oct. 1, 1946, to Oct. 1, 1949, a three-year period. Among these were 204 patients who had neoplasms of the oral cavity and pharynx. Of the tumors of the oral cavity, 167 were carcinoma, which were most frequently found in the tongue and then in the dental ridges, cheeks, and soft and hard palates in the order named. There were 27 sarcomas of the oral cavity. Of the 56 tumors involving the pharynx, 21 were carcinomas and 30 were sarcomas. Among them were 9 carcinomas and 17 sarcomas of the tonsils and 4 carcinomas and 12 sarcomas of the nasopharynx. Thus in the oral cavity carcinoma was much more frequently encountered than sarcoma, but in the pharynx this ratio was reversed, the proportion being 3 sarcomas to 1 carcinoma. These growths occurred most frequently in the ages 40 to 70 years, and the male : female ratio was 3 : 2 for carcinoma and 2 : 1 for sarcoma. These patients first consulted the physician six months to one year after the onset of the initial symptoms.

HARA, Los Angeles.

Larynx

INTERNAL LARYNGOCELE. W. FRANKLIN KEIM and ROBERT G. LIVINGSTONE, Ann. Otol. Rhin. & Laryng. **60**:39 (March) 1951.

Two cases of internal laryngocele with hoarseness and difficulty in breathing are presented, and a surgical approach for the removal of such a laryngocele is described. After tracheotomy, a lateral external approach, with removal of the posterior one-third of the thyroid cartilage and a portion of the hyoid bone when necessary, is deemed best by the authors. Notching of the superior border of the thyroid cartilage facilitates exposure of the neck of the laryngocele sac in some instances. The sac is ligated and amputated close to the laryngeal ventricle. Closure of the wound is performed by usual surgical methods.

While intralaryngeal puncture of the laryngocele may be necessary to afford relief from urgent dyspnea, it is not considered a satisfactory therapeutic measure. Likewise, the use of cautery or partial excision through the laryngoscope is not advisable. Employment of laryngofissure is open to criticism on the basis of excessive intralaryngeal scarring, with consequent impairment of the voice and possible laryngeal stenosis.

STEELE, Kansas City, Kan.

TRACHEOTOMY IN SUBGLOTTICAL LARYNGITIS AND LARYNGOTRACHEOBRONCHITIS. WILLEM WAGEMANN, Med. Klin. **46**:841 (Aug. 3) 1951.

In two boys, 6½ and 5 years old, tracheotomy became unavoidable because of severe acute subglottical laryngitis and tracheobronchitis probably due to influenza infection. The indication for tracheotomy under such circumstances and the postoperative treatment are discussed.

AMBERG, Rochester, Minn. [A. M. A. AM. J. DIS. CHILD.].

Nose

CARCINOMA OCCURRING IN AN ANTROALVEOLAR FISTULA. F. T. HILL and I. I. GOODOF, Ann. Otol. Rhin. & Laryng. **60**:238 (March) 1951.

A case of carcinoma occurring in an antroalveolar fistula following dental extraction and associated with chronic maxillary sinusitis is reported. The patient was found to have epidermoid carcinoma, Grade 3, on biopsy, during exploratory antral surgery. The cervical lymphatics were also involved.

While it is not unusual for malignant changes to develop in a chronically infected sinus, the authors are not aware of any reports of similar changes taking place in an antroalveolar fistula. It is believed that the existence of a chronic inflammatory process, such as is usually present in an antroalveolar fistula, might play a part in the development of malignant change. Therefore, the case illustrates the necessity for early correction of such fistulae.

STEELE, Kansas City, Kan.

SARCOMA OF THE NOSE AND SINUSES. HOWARD C. HIGH, Laryngoscope **61**:803 (Aug.) 1951.

Sarcoma of the nose and sinuses is a comparatively rare condition. Irritation or infection seems to play no etiologic role. Irradiation, however, was the cause in five cases. The condition does not occur so often in the so-called cancer age as it does in younger people. There are no typical symptoms. Symptoms that do occur are due usually to the expansion of the growth and subsequent pressure rather than to invasiveness, as with carcinoma. X-ray examination helps in the diagnosis, but final diagnosis depends upon biopsy. Sarcomas vary greatly, depending upon the amount of fibrous, cartilaginous, myxomatous, and osseous tissue present with a degree of cellularity. The tumors are best eradicated by electrocautery, augmented by radium. The electrocautery seals the blood cells, helping to prevent extension and recurrence. Two unusual cases are reported.

HITSCHLER, Philadelphia.

FRONTAL SINUS: PERMANENT DRAINAGE WITHOUT DEFORMITY. HARRY P. SCHENCK, Laryngoscope **61**:832 (Aug.) 1951.

Despite chemotherapy, there are still a few cases of frontal sinusitis in which surgery is required. However, it is not necessary today to remove nearly as much bone as was formerly necessary. The following technique, which was employed previously only in the absence of bone infection, can now be used in almost all instances.

The incision is made along the line of the eyebrow (previously shaved off) right down to the bone. Digital pressure controls hemorrhage until the bleeding points are secured. If the anterior wall of the frontal sinus is intact, the sinus is entered just below the inner extremity of the eyebrow. A mastoid gouge, a motor-driven burr, or a mallet and chisel are used. The opening may be enlarged with a Kerrison bone forceps, but its extent should be confined to the minimal requirements of the specific situation. The frontal sinus ostium is located by a probe. This is followed by an antrum rasp passed downward to the nasal cavity. The opening is enlarged to accommodate a tube of 10 mm. diameter. A rubber tube is then drawn into the sinus. The tube is guided to curve to the lateral extension of the sinus and is fenestrated. It is often advantageous to remove the anterior portion of the middle turbinate to facilitate these manipulations. After the periosteum is closed, the skin regions are approximated with metal clips, which minimize subsequent scarring. Postoperatively the sinus can be irrigated by passing a No. 8 or 10 soft rubber catheter through the drainage tube. The tube is left in place for six weeks or less, during which time epithelization of the new tract is completed.

A case is reported.

HITSCHLER, Philadelphia.

INTRANASAL MENINGOCELE. T. E. BEYER, J. R. BLAIR, and W. R. LIPSCOMB, Laryngoscope **61**:917 (Sept.) 1951.

Meningoceles are very rare. They result from herniation of the pia and arachnoid through a dehiscence in the skull. Intranasal meningoceles herniate through a dehiscence in the cribri-

form plate. The latter may be confused with polypi. The treatment of choice is ligation of the meningoceles by means of the radical transfrontal ethmoid operation or by craniotomy. Two cases are presented.

HITSCHLER, Philadelphia.

RHINOLOGY IN CHILDREN: RESUMÉ OF AND COMMENTS ON THE LITERATURE FOR 1950. D. E. S. WISHART and J. B. WHALEY, Laryngoscope **61**:957 (Oct.) 1951.

This is the usual fine review of otolaryngology in children. Most important subjects are well covered.

There still is no cure for the common cold. Antihistamines may lessen the early symptoms only, but the course of the cold is unaltered. Some serious side-effects have been noted. The value of constrictors in the treatment of colds is being questioned by more and more authorities.

It is felt that there is an optimum concentration of penicillin, over or under which its value decreases. Streptomycin soon loses its effect because the bacteria quickly become insensitive to it. They do not, however, become insensitive to aureomycin or to chloramphenicol. The former is better for Gram-positive and the latter for Gram-negative bacteria.

Questions concerning sinusitis, the tonsils, allergy, and the use of the nasopharyngeal radium applicator are discussed. Most authorities feel that the standard Crowe applicator, if used properly, is without harm and does much good in selected cases. Several good articles on new growths are reviewed. It is felt that with proper treatment (electrocoagulation and interstitial irradiation) almost all nasopharyngeal fibromas can be nearly or entirely eradicated. This should be done transorally and often is possible in a single operation.

HITSCHLER, Philadelphia.

MYXOMA OF MAXILLARY SINUS. SAMUEL D. GREENFIELD and OREL FRIEDMAN, New York J. Med. **51**:1319 (May 15) 1951.

A 2-year-old white boy was admitted to the Brooklyn Eye and Ear Hospital, Sept. 26, 1949, with a smooth, hard, painless swelling of the left cheek, extending from the left inner canthus to the upper lip. Within the nose there was bulging toward the septum. Adenoids and tonsils were present but not involved. An x-ray showed increased density of the left antrum and absence of dental pathology. The antrum was entered through the canine fossa, which showed pressure erosion. The antrum was completely filled with a grayish-white soft mass, easily detachable and not encapsulated. Bleeding was slight. A simple but complete curettement was done. Walls other than the anterior wall were normal, but the cavity was enlarged to almost adult size by the mass. The cavity was packed with iodoform and the incision closed with nonabsorbable (silk) sutures. Recovery was uneventful and complete after a three-month observation period, wth only slight remaining prominence of the left cheek. The laboratory diagnosis was myxoma of the mucosa of the left antrum. Postoperative irradiation was rejected because large doses of radiation are not well tolerated by a young child, and the results in myoma are not commensurate with the risk. Radical surgery (removal of the superior maxilla) is held in reserve according to the follow-up of the patient. This case is added to the five reported cases of myoma involving the nares and sinuses.

I. W. VOORHEES, New York.

Miscellaneous

A CONCEPT OF ALLERGY AS AUTONOMIC FUNCTION SUGGESTED AS AN IMPROVED WORKING HYPOTHESIS. HENRY L. WILLIAMS, Ann. Otol. Rhin. & Laryng. **60**:122 (March) 1951.

According to Williams, a theory of allergy based on the autonomic vascular reaction is thought to furnish a better working hypothesis from the standpoint of clinical diagnosis and treatment than one based on the antigen-antibody concept.

Allergy may be defined as an inherited predisposition to a localized type of autonomic dysfunction mediated by cholinergic fibers of the autonomic system. There occurs in these localized areas a stereotyped reaction of the peripheral vascular bed consisting of arteriolar spasms with atonic dilatation of the capillaries and venules. This picture, produced only by a maximal stimulus in a normal person, may occur in certain tissues and organs of an allergic person in response to a normally minor stimulus. Such a reaction also may take place as a result of

environmental exposure to stimuli to which a normal person would not react. The same degree of reaction may occur in an allergic person in response to a much less severe stimulus than would be required to produce it in a normal person. These reactions result in a greater or less degree of cellular damage and the release of histamine and other toxic substances, depending upon the type of cell injured.

The clinical picture of allergy may be produced by reaction of the peripheral vascular bed, resulting in the production of anoxic capillary loops, which may lead to typical allergic edema or necrosis and be classified empirically and clinically as allergy. An antigen-antibody reaction may be associated with the vascular reaction and may aid in damaging the cell, but it is a secondary phenomenon, phylogenetically more recent than the vascular component of the autonomic reaction. This definition of allergy includes all types of allergy and yet serves to differentiate clearly allergic and nonallergic processes.

The author has considered this focal type of autonomic dysfunction in a previous paper (Proc. Staff Meet., Mayo Clin. **24**:516 [Sept. 28] 1949). The fundamental assumption was that allergy is a clinical phenomenon. The diagnosis of allergy is primarily made empirically by observation of a gross lesion and of changes occurring in function of organs and tissue in the light of past clinical experience. Observation may be extended secondarily by microscopic and immunologic methods. This is the manner in which the diagnosis of allergy always has been made, but it is inconsistent with a strict adherence to the hypothesis of von Pirquet. Since von Pirquet and, later, Kahn insisted that allergy is altered reactivity, it should be possible to discover the normal prototype from which in each instance allergy has diverged. It would seem reasonable, therefore, to speak of three related but not identical types of allergy: (1) physical allergy; (2) bacterial or tissue allergy, and (3) humoral allergy.

In physical allergy, no antigen-antibody mechanism is present, cellular injury and the typical clinical picture being produced by anoxia. Its normal physiologic prototype may be considered the alarm reaction of Selye.

In bacterial or tissue allergy, the fundamental autonomic (vascular) defense mechanism is retained, but, it is suggested, in the process of phylogenetic development this defense mechanism has been supplemented by the development of protective antibodies. These protective antibodies are primarily attached to certain tissue cells, but circulating antibodies may occur as a sort of byproduct of cellular immunity. The normal prototype of tissue allergy could be considered to be granulomatous inflammation.

In humoral allergy, although antibodies are attached to cells, humoral or circulating antibodies are the outstanding feature. These may be the so-called "blocking" univalent antibodies. It would appear that circulating antibodies take up some of the impact of the invading antigen, so that less severe tissue injury is produced in the host. The normal prototype of humoral allergy could be considered to be suppurative inflammation.

There is nothing in this concept of allergy to suggest that these three types of allergy are mutually exclusive. For instance, perennial vasomotor coryza, which appears to be on the basis of a physical allergy, frequently may be observed, especially in the cold months, and yet have seasonal exacerbations that appear to be on the basis of a specific sensitivity to pollen.

The working hypothesis of allergy as a type of autonomic dysfunction suggests a gradual growth in the animal organism of an increasingly more elaborate defense mechanism and that the new developments are added to, rather than replace, the primitive stereotype autonomic defense mechanism. The concept of allergy as primarily a hyperfunction or dysfunction of this stereotype mechanism does not appear to be in conflict with any of the observed facts. It explains the gradual, rather than the abrupt, transition of one type to the other and why there may be a mingling of types. It explains also why, since the circulating antibody would appear to be a later phylogenetic development, it is not possible to discover evidence of circulating or sensitizing antibodies in so many patients with clinical allergies or to give them symptomatic relief by hyposensitization through the medium of the injection of specific antigens. In the majority of allergic persons, the reaction is not to a type of chemical stimulus (protein) that could be reasonably expected to result in antibody formation. This hypothesis throws the emphasis on the medical and psychosomatic aspect of the treatment of allergy, which is receiving increasing consideration, as opposed to the strictly immunologic aspect.

STEELE, Kansas City, Kan.

Sinobronchitis in Infants and Children. Walter C. Price, J. Pediatrics **38**:590 (April) 1951.

The author has studied the association of chronic sinusitis with chronic bronchial disease. Three hundred eighty-four children were studied. The diagnostic data included history, physical examination, and roentgenograms of the sinuses and the chest. The results were as follows: 1. In a control group of children with no respiratory symptoms 7.7% had roentgenographic evidence of sinobronchitis. 2. Of children with chronic respiratory disease 37.7% had sinobronchitis. 3. Of children with sinobronchitis 46.8% were under 9 years old, and the more severely ill patients in this group were under 5 years old. The author concludes that sinusitis is frequently associated with chronic lower respiratory infection in children.

Macdonald, Pittsburgh [A. M. A. Am. J. Dis. Child.].

Headache and Vertigo Associated with Hypoglycemic Tendency. James A. Harrill, Laryngoscope **61**:138 (Feb.) 1951.

Headache and, to a less extent, vertigo are sometimes related to low blood sugar. The etiologic classification of spontaneous hypoglycemia, as set forth by Conn, applies to patients having such headaches. This classification is given. People who have such headaches practically always have them during the five-hour glucose tolerance test. The headache is dull and throbbing and is often relieved by pressure. A protein diet which gives a steady supply of dextrose over a long period is the most effective treatment. Case reports are included.

Hitschler, Philadelphia.

Bulbar Poliomyelitis: A Respiratory Problem. T. C. Galloway and John Elsen, Laryngoscope **61**:548 (June) 1951.

In bulbar poliomyelitis, secretional obstruction, anoxia, and carbon dioxide accumulation are more deadly than is the direct action of the virus on the vital centers. It follows that treatment of the former gives the better results. Such treatment, not only by Elsen and Galloway but also by others, produced a distinct (two-thirds) lowering of the mortality rate. The lethal damage is caused both by a lack of oxygen and (much more so) by an accumulation of carbon dioxide, which quickly produces a dangerous lowering of the pH. Treatment therefore should not only be directed toward increasing the oxygen but also toward reducing the carbon dioxide concentration.

Secondary effects of secretional obstruction must be looked for. These include congestion, edema, atelectasis, etc., of the airways and lung. Inability to swallow leads to similar trouble. Postural drainage, suction, administration of oxygen, and tracheotomy are included in the therapy of the condition. Maintaining a clear airway is the goal. Food, fluids, and vitamins are administered parenterally meanwhile.

The laryngologist and trained personnel constantly in attendance obviously are necessary.

Hitschler, Philadelphia.

Hemophilus Influenzae Meningitis: Observations on the Treatment of 110 Cases. William G. Crook, B. Reed Clanton, and Horace L. Hodes, Pediatrics **4**:643 (Nov.) 1949.

The results obtained in the treatment of 110 infants and children infected with Hemophilus influenzae are presented, and the various agents used in the therapy of the disease are evaluated. Eighty-seven (79%) survived; sixty-eight (62%) recovered completely. Eight of the 23 deaths occurred within the first 24 hours after hospital admission. Slightly over one-half the children were less than one year old. Only 69% of this group survived, as compared with 90% in the group over one year old; only 50% of the younger group recovered completely, as compared with 75% of the older group.

The plan of therapy underwent various modifications and additions during the eight-year period covered in this study. Forty-four per cent of the patients were treated with specific H. influenzae rabbit antiserum and sulfadiazine; 48% were treated with these agents together with streptomycin (and in many instances penicillin). The remaining 8% were treated with strepto-

mycin alone or in combination with sulfadiazine. The survival and complete recovery rates of those treated with serum, sulfadiazine, and streptomycin were not significantly different from the rates of those treated with serum and sulfadiazine alone. The number of patients treated with streptomycin without serum was too small for analysis.

Specific antiserum used early and in large doses seemed to be a valuable therapeutic agent in these studies. It is believed that this antiserum should be used in the treatment of all patients, except perhaps older children who are only mildly ill.

The high cost of this serum, however, remains a major problem. Streptomycin undoubtedly is a valuable therapeutic agent in the disease; in this study, however, there has been no striking improvement in the over-all results since its introduction.

Sulfonamides and especially sulfadiazine appear to have been of limited but definite value in the treatment of this disease and their continued use in all patients is recommended. Penicillin when given in high dosages would seem to be a helpful therapeutic adjunct when used along with other agents.

Follow-up data on 64 of the 87 surviving patients are presented.

FROM THE AUTHORS' SUMMARY [A. M. A. AM. J. DIS. CHILD.].

NONEPIDEMIC PAROTITIS IN A SERIES COLLECTED FROM AN OTORHINOLOGICAL CLINIC. CARL ADOLF ADNER, Acta otolaryng. **38**:333 (Aug.) 1950.

Disease in the parotid gland may occur by direct spread of infection from the tonsillar area, the external auditory meatus, and the middle ear. Endocrine disease and undernourishment tend to cause hypertrophy of the parotid. As rare complications, pancreatitis and orchitis may supervene. The series here reported is comprised of 24 cases, which is 0.08% of all cases in Adner's department during the period of the study. On the basis of these cases and the total available literature, Adner concludes that in most cases parotitis arises from ductogenic infection. However, poor oral and general condition favor the development of such infections. In five cases in the series the infection was a complication of otitis. Calculi seldom caused infection. Dehydration is a significant causal factor. In some cases the condition was, no doubt, allergic in origin; in some it may be due to imbalance of the nervous system. Treatment is chiefly physical, and not too much reliance is placed upon iodides.

LEWY, Chicago.

Books

Antibiotic Therapy. By Henry Welch, Ph.D., Director, Division of Antibiotics, Food and Drug Administration, Federal Security Agency, U. S. Government; and Charles N. Lewis, M.D., Medical Officer, Director of Antibiotics, Food and Drug Administration, Federal Security Agency, U. S. Government. Foreword by Chester S. Keefer, M.D., Wade Professor of Medicine, Boston University School of Medicine; Chairman, Committee on Medicine, and Chairman, Committee on Chemotherapy of the National Research Council. Price, $10.00. Pp. 562, illustrated by graphs. Indexed. The Arundel Press, Inc., Box 2606, Washington, D. C., 1951.

The sulfonamides ushered in a period of potent chemotherapy which was widely acclaimed until tyrothricin came into being, closely followed by penicillin and a great group of antibiotics which have placed the chemotherapeutics in the background. To many penicillin is still the wonder drug, but its effectiveness is limited largely to the Gram-positive organisms, and therefore it has been superseded in some instances by aureomycin, which is effective against both Gram-negative and Gram-positive organisms. Against certain diseases, syphilis and gonorrhea for example, penicillin is still the principal drug used because of its seemingly selective action in venereal disease.

It is interesting that the word "colds" is not in the index, and the word "respiratory" is also missing, but in general conditions which are "native" to the airways are given proper and sufficient attention in the text. The influenzas (A-virus and the B-virus types) are differentiated; the A is "more likely to cause definite symptoms" than the B, but it is stated that "the value of antibiotics [in influenza] has not been established, their use . . . for the prevention of pneumonia is justified." Two dozen "dosage forms" of penicillin are described, from the crystalline sodium and potassium compounds to the ointment. The general practitioner will be glad to learn that crystalline penicillin in a proper solvent (aerosol or distilled water) for inhalation therapy is helpful in many respiratory conditions, and is remarkably stable in the absence of sterility of the preparation. The crystalline sodium or potassium product has an "expiration date" of 36 months; penicillin procaine is said to be potent for 18 months! This is good news, since contamination, even with proper precautions is probable in the daily round of practice.

The conquest and cure of the meningitides through antibiotics is one of the most brilliant achievements in modern medicine. When antibiotics are administered early enough, and are combined with a sulfonamide, recovery is almost the rule. Twenty years ago, even less, meningitis was commonly fatal; there were few recoveries, since we had no specific medication.

This book should be required reading for every physician, since it brings therapeutic facts in authoritative form and is invaluable clinically.

Directory of Otolaryngological Societies *

INTERNATIONAL

Pan American Association of Oto-Rhino-Laryngology and Broncho-Esophagology

Meeting: Fourth Pan-American Congress of Oto-Rhino-Laryngology and Broncho-Esophagology.
Place: México, D. F., Mexico, Time: January, 1954.
President: Dr. Justo Alonso.
Exec. Secretary: Dr. Chevalier Jackson, 1901 Walnut St., Philadelphia 3, Pa.
President of Congress: Dr. Ricardo Tapia Acuna.

Third Latin-American Congress of Otorhinolaryngology and Bronchoesophagology

Place: Caracas, Venezuela. Time: 1953.

Fifth International Congress of Oto-Rhino-Laryngology

Place: Amsterdam, Netherlands. Time: June 8-15, 1953.
President: Prof. Eelco Huizinga.
General Secretary: W. H. Strubbe, J. J. Viottastraat 1, Amsterdam-Z.
Scientific Secretaries: J. Bijtel and P. G. Gerlings.

NATIONAL

American Medical Association, Scientific Assembly, Section on Laryngology, Otology and Rhinology

Chairman: Dr. Carl H. McCaskey, 20 N. Meridian St., Indianapolis.
Secretary: Dr. Sam H. Sanders, 1089 Madison Ave., Memphis 3.
Place: Chicago. Time: June 11-13, 1952.

American Academy of Ophthalmology and Otolaryngology

President: Dr. Derrick Vail, 700 N. Michigan Avenue, Chicago 11.
Executive Secretary-Treasurer: Dr. William L. Benedict, 100-1st Ave. Bldg., Rochester, Minn.
Place: Palmer House, Chicago.

American Board of Otolaryngology

Secretary: Dr. Dean M. Lierle, University Hospitals, Iowa City.
Place: Palmer House, Chicago. Time: Oct. 6-10, 1952.
Place: Royal York Hotel, Toronto, Canada.

American Broncho-Esophagological Association

President: Dr. Herman J. Moersch, Mayo Clinic, Rochester, Minn.
Secretary: Dr. Edwin N. Broyles, 1100 N. Charles St., Baltimore 1.
Place: Royal York Hotel, Toronto, Canada.

American Laryngological Association

President: Dr. H. Marshall Taylor, 111 W. Adams St., Jacksonville, Fla.
Secretary: Dr. Louis H. Clerf, 128 S. 10th St., Philadelphia 7.
Place: Royal York Hotel, Toronto, Canada.

American Laryngological, Rhinological and Otological Society, Inc.

President: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Secretary: Dr. C. Stewart Nash, 708 Medical Arts Bldg., Rochester 7, N. Y.
Place: Royal York Hotel, Toronto, Canada.

* Secretaries of societies are requested to furnish the information necessary to keep this list up to date.

SECTIONS:

Eastern.—Chairman: Dr. Francis W. Davison, Geisinger Memorial Hospital, Danville, Pa.
Place: Waldorf-Astoria, New York.
Southern.—Chairman: Dr. Lester A. Brown, 490 Peachtree St., Atlanta.
Place: Academy of Medicine, Atlanta.
Middle.—Chairman: Dr. Ralph J. McQuiston, 20 N. Meridian St., Indianapolis.
Place: Indianapolis Athletic Club, Indianapolis.
Western.—Chairman: Dr. Howard P. House, 1136 W. 6th St., Los Angeles.
Place: County Medical Building, Los Angeles.

American Otological Society

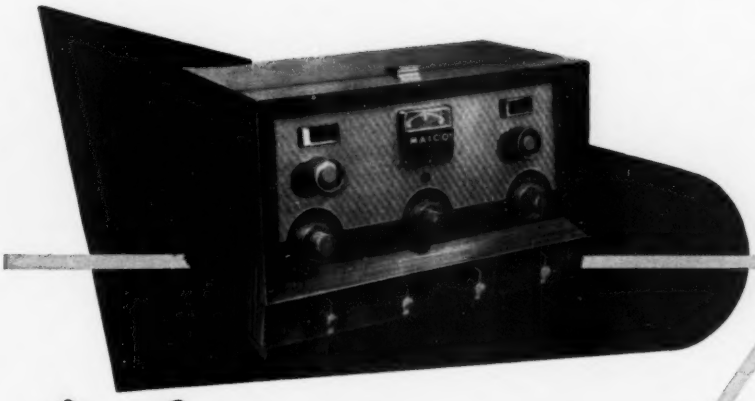
President: Dr. Gordon D. Hoople, 1100 E. Genesee St., Syracuse, N. Y.
Secretary: Dr. John R. Lindsay, 950 E. 59th St., Chicago.
Place: Royal York Hotel, Toronto, Canada.

American Otorhinologic Society for the Advancement of Plastic and Reconstructive Surgery, Inc.

President: Dr. Benjamin H. Shuster, 1824 Pine St., Philadelphia.
Secretary: Dr. Louis Joel Feit, 50 Park Ave., New York 16.

American Society of Ophthalmologic and Otolaryngologic Allergy

President: Dr. William H. Evans, 24 Wick Ave., Youngstown 3, Ohio.
Secretary-Treasurer: Dr. Joseph Hampsey, 806 May Bldg., Pittsburgh 22.



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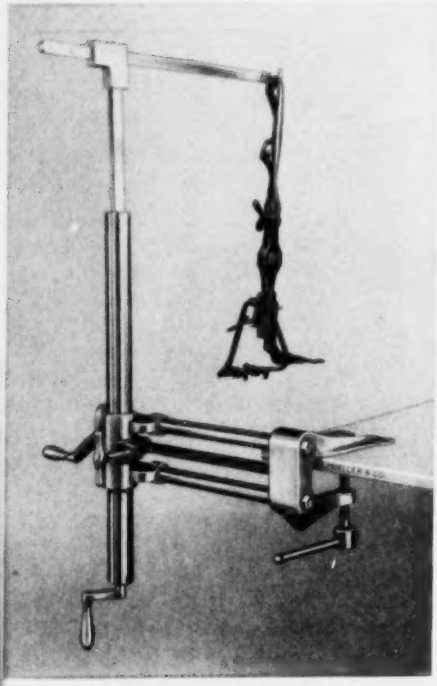

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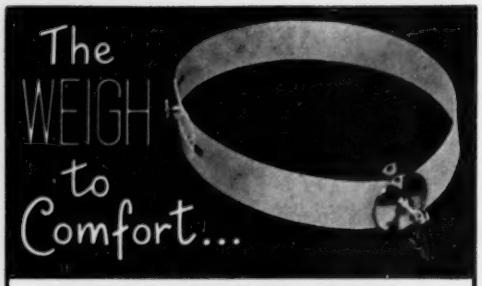

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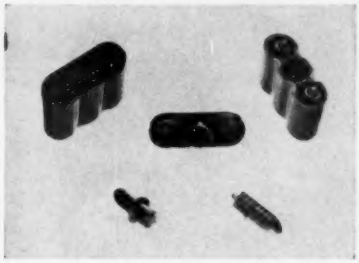

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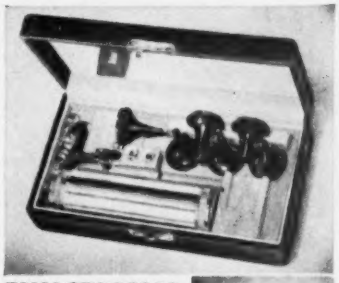

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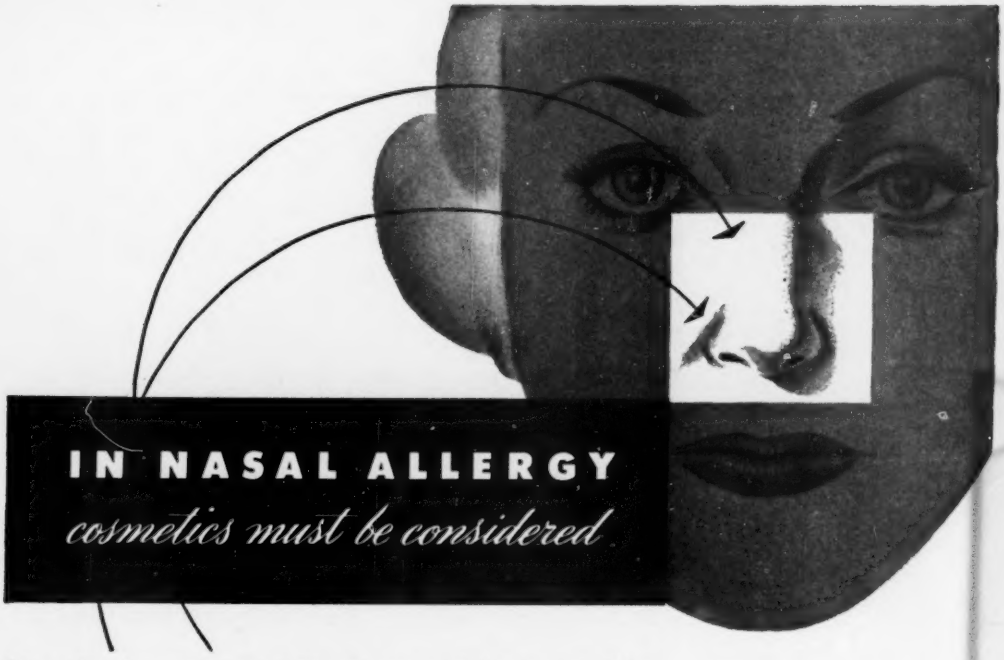

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1. Tuft, L.: Clinical Allergy. Philadelphia, W. B. Saunders Co., 1947, pp. 335-336.
2. Hansel, F. K.: Allergy of the Nose and Paranasal Sinuses. St. Louis, C. V. Mosby Co., 1936, p. 769.
3. Kelley, S. F.: Choice of Sympathomimetic Amines. Cornell Conferences on Therapy, II, 1947, p. 156.

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